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WOBURN CHILDHOOD LEUKEMIA FOLLOW-UP STUDY

Volume II
Appendices

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FINAL REPORT

July 1997

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
BUREAU OF ENVIRONMENTAL HEALTH ASSESSMENT

WOBURN CHILDHOOD LEUKEMIA FOLLOW-UP STUDY

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APPENDIX I

Review of the Literature

ADDITIONAL INFORMATION FROM THE

SCIENTIFIC LITERATURE

Descriptive Epidemiology of Childhood Leukemia

Age and Sex

AML occurs at a constant low rate throughout early childhood, the annual incidence rising sharply between the mid-teens and mid-twenties to about five to ten cases per million. ALL shows a strikingly different pattern; peak incidence (around 40 cases per million [Linet, 1985]) occurs between ages one and four, and incidence decreases rapidly to a low of only about one case per million population annually at around age 30 [Linet, 1985].

Data are mounting to suggest that leukemia occurring in children under seven years of age is a disease distinct from that affecting older children and adults. As Lancet editors stressed in a recently published editorial [1990], early childhood cases of ALL tend to be the B-cell type, whereas leukemia affecting older young people is more heterogeneous with respect to subtype. Alexander and McKinney [1990] have further pointed out that there is little correlation in British statistics between county standardized mortality ratios for leukemia occurring at ages one to seven and that occurring at ages eight to 24 -- the suggestion being that the early childhood form is also etiologically different from that affecting older children. As will be seen from the paragraphs that follow, many inconsistencies have emerged from studies of leukemia in general and of childhood

leukemia in particular. This inconsistency may have resulted, in part, from a failure to treat childhood and adult leukemias as separate entities and from the lack of a uniformly applied definition of childhood in those studies that have either concentrated on children or reported results separately for children and adults.

Socioeconomic Status (SES)

Van Steensel-Moll et al. [1986] found that Dutch children affected with leukemia had a lower incidence than controls of serious infections during the first year of life, although cases and controls were similar in terms of common-infection rates. The authors were surprised by these results and considered selective survival to be an unlikely explanation for this phenomenon in a developed society. It could be argued, however, that these findings might have been predicted by Knox et al. [1984], who spoke of "pre-emptively fatal hazards, such as pneumonia." To elaborate, while we might not expect even a pre-leukemic child to die of a simple cold or other common infection in a western society, it would not seem unreasonable that pneumonia would be more likely to have a fatal outcome in such children as compared with normal children. The consequence of this selection would be a decreased ability to find victims of childhood leukemia who had survived pneumonia. Nevertheless, Van Steensel-Moll et al. raise the possibility that infection may have a protective effect against leukemia. In support of this

idea, Van Steensel-Moll et al. cite data indicating that, as countries improve socioeconomically (a change that is generally accompanied by a decrease in the infection load), the rate of childhood lymphoma drops, but the rate of leukemia increases. These data could, however, reflect a directly causal role for infectious agents in lymphoma etiology and a pre-emptive effect of exposure to these agents in childhood leukemia occurrence.

Urban/Rural Environment, Population Density and/or Growth

When Linet published her review of leukemia epidemiology in 1985, few data were available with which to address the question of whether there was any relationship between degree of urbanization and leukemia incidence. Donham et al. [1980], using data from the Third National Cancer Survey, found that, for males in Iowa, ALL rates were higher in rural areas versus urban ones, but no such difference was found for females. No urban/rural differences emerged for males from analyses performed by the International Agency for Research on Cancer (IARC), and these data suggested that urban females have higher leukemia rates than do rural females. Linet [1985] suggested that the different findings from the two data sets might be explained by the lack of separation of cell types in the IARC data or by differences in the conditions of rural life in Iowa versus other locations. In any case, these analyses did not pertain specifically to childhood leukemia. Data analyses recently completed by Alexander [1990a] suggested that the positive association noted

in various data sets between childhood leukemia incidence and rural isolation was stronger in young children versus older ones.

Several more analyses pertaining to this and associated factors in relation to childhood leukemia incidence have been conducted in recent years. The impetus for the additional research was the finding of high leukemia rates near nuclear-fuel-reprocessing plants in Great Britain. Kinlen [1988] pointed out that the areas with the plants and the childhood leukemia clusters were rural and characterized by rapid population growth -- the implication being that the rural character of the areas and their rapid change to more populous communities was directly linked to the leukemia excesses and that the location of the nuclear facilities in the areas was merely coincidental. Kinlen et al.'s studies of this hypothesis focused on new towns that have sprung up in rural areas of Great Britain in response to the development of a new industry. Alexander and McKinney [1990], however, claim that the explanation for the increases is community isolation, not population mixing; to support their claim, these authors point to their observation that, in developed societies, the age peak in childhood leukemia incidence (manifested as high incidence in the one-to-seven years age range) is more pronounced in rural (regardless of population growth) and low-SES communities as compared to urban and high-SES areas. Furthermore, these authors and others [Alexander et al., 1990a] failed to find evidence of an association between childhood ALL incidence and commuting-to-work distances, but the

descriptive data they used did support the hypothesis that childhood leukemia incidence is related to distance from built-up areas.

Langford and Bentham [1990], on the other hand, favor the hypothesis that rapid population growth (regardless of the original character of the areas) explains the excess childhood leukemia observed in the nuclear-plant- containing communities and other isolated towns. These authors found excesses of childhood leukemia in areas of England and Wales whose populations had increased by more than 50 percent between 1961 and 1971. Excesses were not observed for areas that had experienced smaller population increases.

As a final note on this subject, it is useful to consider Alexander and McKinney's caution that the community characteristics that are related to leukemia occurrence during the peak-incidence years in childhood may have no etiologic significance.

Secular Trends

Mortality from childhood leukemia increased dramatically in England and Wales between 1911 and 1955, an era during which the disease was uniformly fatal. Doll [1989] attributes the increase to either heightened incidence or improved diagnostic capability. In the Discussion of his paper, he also invokes Stewart and Kneale's now well-known explanation involving the deaths of large numbers of undiagnosed leukemias from infections to which, as

pre-leukemics, they would have been unusually susceptible and for which powerful treatments were unavailable until close to the time of peak leukemia mortality. Mortality from childhood leukemia decreased rapidly in recent years due to treatment advances.

Clustering

Small clusters of rare events constitute a natural feature of random distribution, necessitating the observation of some minimal degree of clustering to define a meaningful group of cases and raising the possibility that childhood leukemia cases only appear to cluster because of the extreme inaccessibility of even small numbers of cases.

Another difficulty that has been encountered in cluster identification pertains to the acceptable parameters of the epidemic curve for cancer. Critics of cluster identifiers have often pointed out that if a longer time period were considered, incidence would appear more normal, because clusters often occur between periods of low incidence. In Woburn, Massachusetts, for example, 21 cases of childhood leukemia occurred between 1969 and 1986, but no cases have occurred since the spring of 1986.

Modern tests of clustering have involved the apriori specification of a place and time period and the search for evidence of clustering among all cases that have arisen within those pre-specified parameters. After reviewing the cluster analyses that had been completed by 1976, Smith [1982] concluded

that the data provided ". . . very little support for the notion that space-time clustering of leukemia is a feature that etiologic hypotheses must encompass."

In a more recently published paper, Doll [1989] pointed out that, because the statistical techniques that have been applied to the clustering phenomenon have been relatively weak, the question should be considered still open.

Interest in the clustering of childhood leukemia was re-awakened recently by reports of clusters' occurring in the vicinity of Great Britain's nuclear-fuel-reprocessing facilities. Much of the criticism that has been launched against those who have hypothesized a causal role for radioactivity in this clustering has centered on the question of whether such clusters might have arisen by chance.

Some authors of recently published reports have presented evidence supportive of childhood leukemia clustering in Great Britain [Morris, 1990; Alexander et al., 1990b; Knox and Gilman, 1992a,b]. Studies conducted by Knox and Gilman [1992a,b] concerned all of Great Britain; Alexander et al. [1990b] specifically targeted for cluster analysis an area containing a nuclear-reprocessing facility in which a well-known anecdotal cluster was located; and Muir et al. [1990] purposely singled out an area lacking a nuclear facility. The last-mentioned team of investigators found individual clusters but concluded that the tendency of cases of childhood cancer to cluster did not exceed that expected by chance alone. All of the other investigators

found evidence of clustering, but certain complexities emerged in the data. For example, although Knox and Gilman [1992a] demonstrated both space-time and geographical clustering, they found no tendency for cases to cluster in families or even in monozygotic twin pairs -- a result that is difficult to reconcile with any etiologic hypothesis, given the necessity of the environmental agents responsible for the clustered diagnoses to rarely affect siblings and virtually never affect both members of a twin pair. Both Knox and Gilman [1992a] and Morris [1990] favor a promotional role for the cause of the clustering phenomenon, and both teams consider an infectious agent to be a likely candidate. Vivian Morris [1990], in fact, tested the hypothesis that cases in close pairs had different infectious-disease profiles from matched controls. She found some evidence that children involved in close pairs who developed cancer relatively late had lower previous-infection rates than their matched controls. The resemblance between Morris' results and those of Van Steensel-Moll [1986] discussed in a previous section is noteworthy. Like Van Steensel-Moll's findings, Morris' results seem consistent with the idea that high infection rates would be difficult to find in children diagnosed at older ages, because pre-initiated children who had experienced frequent or serious infections might have either died of infection before leukemia was diagnosed or been diagnosed earlier.

Doll's recent review [1989] provides a scenario (which Doll attributes to Greaves) that might explain some of the complexities of clustering noted above. Greaves' [1988] idea of childhood leukemia initiation involves two separate mutations -- one occurring in fetal life and one occurring later. Both events are seen by Greaves as random DNA-transcription errors whose likelihood is increased by the high rates of cell division that prevail when the B cell precursors are multiplying rapidly and when antibody-producing cells proliferate in response to antigens encountered by the child in the environment outside the uterus. In such a scenario, an identical twin of a potential leukemic would be no more susceptible than an unrelated individual whose cells were undergoing the same changes at the same time. Clustered diagnoses might occur in relation to infectious-disease epidemics not because the infectious agents have a directly causal role but because they provide the circumstances that increase the likelihood of the occurrence of a random event. As Doll points out, such a mechanism would not necessarily preclude a role for environmental factors. Also explained by Greaves' model is the predominance of the "B" cell type of ALL and the sharply focused age distribution of the disease. Greaves claims that animal data suggest the existence of an immune-stimulus-initiated positive feedback mechanism controlling B-cell proliferation which is not detectable in the thymus, where "T" cells originate. The age peak corresponds to the typical timing of the first major immunologic challenge presented to the young

child. Leukemia cases arising in older children might be explained by delayed occurrence of immunologic challenge or, perhaps, by some other mutagenic or proliferative stress resulting in a second mutation in children whose first mutation occurred during the early childhood period of high immunologic challenge.

Alexander [1992] recently found evidence of spatial clustering near the time of birth of children who developed leukemia late in childhood. The evidence of clustering was weaker for children whose disease manifested itself during the early childhood peak. Although the immunophenotypes of the cases were not known, Alexander reasoned that the older cases may have been T-cell ALL, which is more common than B-cell ALL after the early childhood peak, and that clustering may be a feature of the epidemiology of T-cell ALL only.

Etiologic Hypotheses

A Lancet editorial [1990] recently proclaimed that the only known causes of leukemia in children are exposure to X-rays in utero and ionizing radiation. Furthermore, Knox et al. [1984] believe that most of the epidemiological associations described in the previous sections ". . . are best interpreted in non-causal terms . . . ." This is not to suggest that all childhood leukemia is caused by radiation; according to Lancet editors, the identified causes are responsible for no more than eight percent of cases, with the remaining cases unexplained. In

1987, Cartwright listed unusual patterns of infection, lack of vaccination, and chloramphenicol use as the only other known leukemia correlates with etiologic potential.

Our understanding of the causes of childhood leukemia is hampered by the historical treatment of all of leukemia as a single disease. Linet's 1985 review of the leukemias sought to characterize as fully a possible the epidemiology of the different cell types. Even Linet, however, failed to distinguish childhood leukemia from adult forms. The body of epidemiologic studies on childhood leukemia has been growing in recent years, however. Researchers have concentrated on parents' histories of chemical and/or radiation exposure, the possible role of infectious agents, the effect of mothers' cigarette smoking during pregnancy and of passive smoking during childhood, the effects of exposure to electric and magnetic fields, and the possible etiologic significance of residing near nuclear facilities.

Infectious Disease

The attempt to relate infectious agents to leukemia has a long history. The tendency to blame childhood leukemia, in particular, on viruses is, in fact, so strong that attempts have been made to explain most of the epidemiologic features discussed above in terms of the principles of infectious disease epidemiology. The associations noted between childhood leukemia and low birth order [MacMahon, 1992a], high socioeconomic status

[Van Steensel-Moll et al., 1986], and rural environment [Kinlen, 1988], for example, have been hypothesized to be virally mediated -- in all three cases, by delayed or reduced exposure to common viruses.

As noted above, the perceived tendency for childhood leukemia to cluster has provided some of the impetus behind the search for an infectious cause of childhood leukemia, but there are other reasons to suspect that the disease might have a viral etiology, at least in some cases. Among these is the certainty of the role of viruses in animal leukemia [Heath, 1982]. Retroviruses, which contain RNA as their genetic material [Davis et al., 1980], cause leukemia in cows, cats, chickens, gibbons, birds, and primates. The viruses can be transmitted in various bodily fluids, and leukemia follows infection by months or years [Linet, 1985].

The observation that leukemia viruses cross species [Linet, 1985] barriers has perhaps fueled the search for evidence of transmission of animal leukemia viruses to humans. This particular subject has received considerable attention in the literature, without any hint of an association, but Kranz [1983], who has reviewed much of the research, concludes that the studies were unlikely to have revealed any relationship had one existed. The main problem with the past investigations concerned the vagueness of exposure assessment -- a weakness derived from ignorance about what circumstances might be required for transmission (e.g., contact with a sick animal, contact with an

animal with leukemia, intimate contact, long-term association, long latency between association and outcome, etc.).

Linet [1985] refers to ecologic studies showing correlations between human leukemia incidence or mortality rates and rates of leukemia in cows. She reports that some such studies have revealed associations and some have not. Even if significant associations had been found in the majority of such investigations, this would constitute only weak evidence for an infective cause of leukemia or animal-to-human transfer of the microbe -- the weakness of these data deriving from the failure of ecologic designs to ascribe exposure to individuals (i.e., Did the people who developed leukemia come in contact with cows?) and to eliminate alternative explanations for the increased leukemia incidence occurring at the time of bovine epidemics. Individual-level investigations have relied on similarly poor surrogates for exposure to animal viruses such as an entry of "farming" for occupation on death certificates.

Strongly implicating viruses in the etiology of childhood leukemia is the association of a retrovirus with a form of adult leukemia (ATL = adult T-cell leukemia) which has been identified in Japan, the Caribbean Basin, and Central Africa [Kazura et al., 1987].

Authors of recently published papers have asserted that ATL is caused by a latent infection with human T-cell leukemia virus type 1 [Kushuhara et al., 1987; Reeves et al., 1988; Kazura et al., 1987]. The basis for this claim appears to be the repeated

demonstration of the virus in the genome of lymphoblastoid cells (but not other cells) of ATL victims. The absence of the virus from all but neoplastic cells shows that it is acquired rather than inherited [Robert-Guroff et al., 1982]. Antibodies to the virus have also been demonstrated in samples from patients with ALL, CML in blast crisis, and acute monocytic leukemia (but not other AML types or CLL), but the seropositive proportion of non-ATL samples was considerably lower than that for ATL samples.

Some interesting data were obtained by Izui et al., who compared the single-stranded-DNA-binding activity of sera obtained from patients with ALL, AML, and CML to that from controls. The finding of higher levels in cases versus controls would be significant, because single-stranded DNA, which does not occur in humans, results from an enzyme-mediated process in RNA viruses; furthermore, the infection of humans by such viruses should elicit the production of antibodies capable of binding to the single-stranded DNA.

The levels of single-stranded-DNA-binding activity were higher in all three types of leukemia patients versus controls. Of course, it is unlikely that all RNA viruses cause leukemia, such that the presence of the antibodies in a leukemia patient does not prove that a virus caused the leukemia. On the other hand, the comparatively higher activity in cases versus controls in a fair-sized study (175 cases) is certainly suggestive.

The notion that leukemia might be a rare response to a common virus has been investigated in a number of studies

attempting to relate increases in leukemia incidence to epidemics of common infections. Heath [1982] contends that enough studies of influenza have been conducted to remove the flu virus from suspicion, but the potential role of varicella virus is still under investigation.

Linet [1985] reports on a case-control study completed by Gharton et al. in 1971 in which measurements of antibodies to various common viruses revealed that only one case-control difference -- the proportion of cases positive for herpes virus was lower than the control proportion. As Linet points out, these results might indicate that the leukemics were less able to mount an immune response against herpes virus and, as a consequence, sustained more than the usual degree of harm from this common pathogen.

Although the identification of anecdotal clusters of childhood leukemia cases have been used as circumstantial evidence of an infectious etiology, specific searches for clustered occurrence of cases have been unsuccessful until recently [MacMahon, 1992b]. Alexander et al. [1992] demonstrated via a case-control design that childhood victims of ALL were more likely to have had cases than controls as nearest neighbors between birth and diagnosis. This difference was highly statistically significant and was attributable mainly to the effect noted for children under age five at diagnosis. Interestingly, the nearest-neighbor cases of the young (age 0 - 4 years) leukemic children tended to be older (age 5 - 14 years)

children. Furthermore, the majority of affected children with nearest neighbors who were also cases had been neighbors for a period that extended back to more than two years prior to the diagnosis of at least one of the two children. The authors also found evidence of linkage of groups of younger cases around a smaller number of older neighbor cases. The results led to the following three hypotheses relating to an unknown infectious agent, Z [Alexander, 1992]:

I. Some children become persistently infected following exposure in utero or around the time of birth. These children have increased risk of developing leukemia, especially ALL at older age of onset (5 years or older).

II. Post-natal exposure to Z may increase the risk of ALL in young children (aged under 5 years at diagnosis) and in particular

III. ALL in the 'childhood' peak (ages 2 - 4 years) may be a rare sequelae of recent first exposure to Z [1992, p. 589].

Alexander et al. see parallels between hypothesis III. above and a model of common childhood ALL causation proposed by Greaves, a theorist who envisions a role for viruses which allows for interaction with other environmental factors. Greaves claims that the traditional sequence of events in terms of the timing of the introduction of microorganisms protects against common childhood ALL, and that such factors as decreasing family size

and a trend away from breast feeding have contributed to (1) secular increases in leukemia incidence and (2) an association between leukemia incidence and SES.

In Greaves' view, the viruses which function in the etiology of common childhood leukemia are not transforming agents but promoters stimulating the division of already transformed cells. As explained above, the initial mutation, according to Greaves' model, arises in fetal life as a consequence of the high spontaneous mutation rate and proliferative stress which are believed necessary for the development of immune competence. Alternatively, this period of high proliferation (and only limited DNA-repair capability) could be a time of increased susceptibility to environmental mutagens such as radiation. The model predicts that the resultant clone of transformed B cells will lie dormant until the onset of the immune response, when the introduction of microorganisms stimulates a second period of enhanced proliferation leading to a second mutation in the preexisting transformed clone.

Kinlen, who also espouses the idea that common infections figure in leukemia incidence, has attempted to explain the specific childhood leukemia clusters which have been observed around nuclear plants in Great Britain [Mathews, 1988] by invoking an infectious origin. Kinlen's theory, put forth in 1988, centers around the isolation of the nuclear facilities from population centers.

Propagated epidemics (which spread by person-to-person contact) are prevented in sparsely populated areas by the low likelihood that an affected individual will come into contact with a susceptible one before the period of communicability lapses. When small populations are increased all at once by the influx of large numbers of people (as when a nuclear power plant newly built in an isolated area creates job opportunities), epidemics may occur through the introduction of new viruses and through increased probability of person-to-person contact. Eventually, herd immunity -- a phenomenon which prevents epidemics through a mechanism similar to that described above -- takes over.

Kinlen claims that the towns of Thurso in Scotland and Seascale in England, where leukemia clusters have been noted, have in common not only a nuclear facility but also a history of isolation prior to the building of a plant and a large population increase thereafter.

The hypothesis that this history, and not the plant, has caused leukemia via a viral mechanism requires a test in an area with a similar past but with no nuclear facility to complicate matters.

Kinlen could find only one town in all of England and Wales with suitable characteristics, and his calculations of leukemia mortality ratios revealed significant excesses in Glenrothes during the period 1951 - 1967. Cartwright et al. [1989] found several points to criticize in Kinlen's paper, including his use

of (1) questionable estimates of the sizes of historical populations, (2) a comparison town which may not have been isolated, and (3) mortality rather than incidence data. The most obvious of the paper's problems, though, is the failure of the author's methods to find any evidence of significantly elevated leukemia mortality in Thurso (the high-leukemia area that Glenrothes was selected to match) -- a surprise which Kinlen doesn't even mention. Wakeford [1989], however, points out that the sizes of the two populations were quite different during the period of interest, such that, if confidence intervals had been constructed around the mortality ratios, the data from the two towns would have been compatible, and the estimate for Thurso could have been interpreted as consistent with a large leukemia excess. In papers published more recently by Kinlen and his colleagues [1991, 1993], associations were described between childhood leukemia and commuting distance, the proportion of the population commuting to work, and other indices of increased opportunity for making new social contacts (features hypothesized _apriori_ by Kinlen to have a similar effect on herd immunity as the creation of a new town).

Interestingly, the Glenrothes childhood leukemia excess was limited to children under five years of age -- a detail that Kinlen has tried to incorporate into his infectious-disease-etiology hypothesis by suggesting that the deficit noted at older ages reflects an immunizing effect of limited exposure to a specific infectious agent or agents. Alexander and McKinney

[1990], however, reminded Lancet readers of other data indicating different etiologies for leukemia in the childhood peak and at older ages, their point being that the epidemiology of leukemia in older children need not resemble that in younger children for a particular explanation of the latter cases to be valid. In particular, Alexander [1992] provided strong evidence of the clustering of birth addresses of children diagnosed with leukemia between the ages of five and 15 and clustering of addresses occupied closer to the time of diagnosis for children diagnosed at earlier ages (It should be noted, here, however, that Morris [1990] found the opposite.). The author's interpretation of the former phenomenon in terms of an infectious etiology rests on the length of time between clustering and diagnosis. For common, say, pollutant exposure near the time of birth to be the cause of the older-diagnosed cases, Alexander contends that an unusually long latent period must be assumed. The association found by Stewart and Kneale [1970] between in utero X-ray exposure and leukemia at ages ten to 15 should be kept in mind here, though, as should the connection between adult vaginal cancer and in utero diethylstilbestrol exposure [Savitz et al., 1990]. In any case, Alexander considers the long-latency assumption an unnecessary complication for an infectious etiology if persistent infection is set up close to the time of birth, allowing the resultant malignant changes to occur any time thereafter.

Adding to the attractiveness of Alexander and McKinney's ideas is the fact that the viruses that cause leukemia in animals

do ". . . persist in the host and induce disease many months or years . . . ." after infection [Lancet editors, 1990]. Furthermore, Lancet editors [1990] claim that age at infection is an important determinant of persistence in both feline leukemia and virally mediated human liver cancer.

MacMahon [1992b] recently reviewed more newly published papers authorized by Kinlen, each designed to test his social-isolation-followed-by-population- mixing hypothesis with the risk factor variously parameterized in descriptive- level studies. Results were positive but weak for New Towns other than Glenrothes [Kinlen, 1990], and for British counties where high proportions of military servicemen had been stationed. An association was also found between childhood leukemia incidence and extent of commuting increase [Kinlen et.al., 1991]. The fact that this latter phenomenon would bring together previously isolated adults, not children, was taken as supportive of the hypothesis that contacts between adults may influence the incidence of leukemia in children. Most recently, Kinlen et al. [1993a] have considered the incidence of childhood leukemia and non-Hodgkin's lymphoma in areas of Scotland with various proportions of the working population employed by the oil industry. The theory was that, inasmuch as oil terminals represent extreme examples of sites where mixing has occurred among men culled from widely separated rural and urban locations, any effect of mixing of adults on childhood leukemia should be demonstrable in the rural towns from which the oil workers were

hired. Interestingly, where associations were found (only for brief periods in the early post-mixing period and only in areas with a high proportion of workers employed in the oil industry), the fathers of the leukemic children were almost never employed in the oil industry, a finding that would argue against any effect of the occupation, itself, on the fathers that is later manifested in their children (the mechanism that has been invoked to explain the tendency of fathers of leukemic children living near the Sellafield nuclear-fuel reprocessing plant to have been employed in the nuclear industry and monitored for radiation [Gardner et al., 1990]). Although MacMahon found several inconsistencies in the data to comment on, he noted that the hypothesis was becoming more focused as a result of the several studies. What is impressive is the merging, recently, of the hypotheses of the different research teams headed by Kinlen, Greaves, Alexander, and Knox. While Greaves and Alexander initially considered the timing of virus introduction, in general, to be the key factor, newly amassed evidence of clustering is shifting their thinking towards Kinlen's idea that some childhood-leukemia cases represent unusual responses to a specific virus. Furthermore, all the researchers now seem to espouse the notion that the geographical clustering of some diagnoses may result from reinfection, and Kinlen now seems to share Alexander's view that leukemia in older children may arise from a double dose of virus, the first introduced before or shortly after birth, when it establishes a persistent infection,

and the second introduced close to the time of leukemia diagnosis. Also generally palatable now is the notion that delayed introduction to infectious agents or to a specific virus may mediate rare sequelae.

While it is challenging to arrive at a theoretical framework that can completely account for the epidemiology, the following represents this author's attempt to find a model that best fits all of the data: Suppose that viruses, and possibly a single virus, play a role in both B cell and T cell childhood ALL. Suppose further that the virus operates somewhat differently in the two forms.

In the T cell subtype (affecting older children), the virus might actually cause leukemia by setting up a persistent infection around the time of birth such that reinfection with the same virus near the time of diagnosis has a promoting effect on an already transformed clone of cells.

In children who will develop B cell ALL in the childhood peak, transformation of B cells would have occurred by chance during the prenatal period of proliferative stress discussed by Greaves. To accommodate Knox and Gilman's monozygotic twin data, the mutation must occur after the stage in fetal life at which the two twins separate. Children in whom cell transformation has occurred may or may not develop leukemia; however, the probability might be increased in those that experience delayed introduction to either viruses in general or a specific virus.

Such a delay could be effected through any or all of the means suggested by Greaves and Kinlen.

A striking feature of the epidemiology of childhood leukemia is the absence of strong evidence of affected children directly infecting each other. Of note, here, is Knox and Gilman's [1992a] failure to demonstrate twin concordance. Most studies, however, would not have been capable of elucidating the pattern described by Alexander (i.e., younger cases clustered around an older one).

In attempting to make sense of the many theories and studies related to a hypothesized infectious etiology for childhood leukemia, it is good to keep in mind MacMahon's reminder that, in the search for a cause of testicular cancer, the epidemiology of which shares some features with that of ALL, researchers have been ". . . more inclined to think of explanations in terms of hormones than viruses . . . ." The fact is that the focus on the infectious-etiology hypothesis has done more to elucidate ALL's natural history and to emphasize probable etiologic differences among the various forms than it has to demonstrate the theory's validity. No data have eliminated the possibility that the initial transformation that seems to happen during fetal life in those children diagnosed during the childhood-peak years might sometimes be caused by a carcinogenic exposure.

Genetic Predisposition

Familial aggregation of leukemia -- a phenomenon which could result from inheritance or from a shared environment -- is known [Linet, 1985; Heath, 1982; Sandler and Collman, 1987] -- usually with a single cell type occurring in all affected family members. Investigators have generally failed, however, to find blood-cell or other markers that typify affected members of leukemia-prone families [Linet, 1985]. Heath [1982] reports that numerous surveys have considered the question of familiarity without consistent evidence of more than faintly increased risk to close relatives of leukemics. This author maintains that familial aggregation, if it does exist, seems to involve mainly CLL, which does not occur in childhood.

The concordance of twins with respect to leukemia incidence is of interest to some in the determination of the role of genetics in etiology. Identical, or monozygotic (MZ), twins come from a single fertilized egg; the two fetuses thus have the same genetic make-up. This would not be true for fraternal, or dizygous (DZ), twins.

Twins have been used in the past to shed light on nature-versus-
nurture issues. For example, if identical twins reared apart both develop a psychosis, then the conclusion of a genetic basis is supported. A complicating factor for leukemia, however, is the known tendency for <u>in utero</u> exposure to at least X-rays to cause childhood leukemia [Heath, 1982; Sandler and Collman, 1987;

Greaves and Chan, 1986; Sztanyik, 1978]. Consequently, both MZ and DZ twins should show greater concordance than other sibships, even if inherited defects are unimportant. Assuming that MZ twins do show higher levels of concordance for leukemia than do DZ twins, the explanation could lie in the twins' shared circulatory systems rather than their identical genes (i.e., some members of a clone of leukemic cells produced or arising spontaneously in one fetus could take up residence in the other), and this seems to be the prevailing interpretation of the phenomenon [Heath, 1982; Sandler and Collman, 1987; Greaves and Chan, 1986]. The studies of childhood leukemia clustering recently completed by Knox and Gilman did not support the hypothesis that MZ twins tend to be concordant for leukemia.

Features of leukemia epidemiology which do tend to foster the notion that either the disease itself or a predisposition to it can be inherited include its status as one of a very few cancers that occurs in childhood, and particularly in early childhood, and observed associations between certain hereditary, or at least congenital, abnormalities and increased incidence of leukemia. It is undoubtedly significant that (1) all such syndromes involve abnormal numbers of chromosomes (e.g., Down's syndrome, Kleinfelter's syndrome) or greater-than-normal fragility of chromosomes (e.g., Bloom's syndrome, Fanconi's anemia) [Greenwald and Greenwald, 1983; Heath, 1982, Sandler and Collman, 1987; Linet, 1985; Page and Asire, 1985], (2) broken or visibly abnormal chromosomes are characteristic of the leukemic

state [Heath, 1982; Linet, 1985; Sandler and Collman, 1987; Miller et al., 1986], (3) known leukemogenic agents such as ionizing radiation and benzene cause chromosomes to break and/or combine with one another in unusual ways [Littlefield et al., 1980; Upton, 1982; Littlefield and Joiner, 1978], and (4) genetic mutation has long been hypothesized to constitute one or even the only mechanism of carcinogenesis. Sandler and Collman [1987] add that there is strong concordance between the break-points observed for chromosome translocations and deletions in leukemia and the location of oncogenes. Nevertheless, it is important to recognize that the inherited and/or congenital syndromes mentioned above make a very small contribution to the total incidence of leukemia [Greaves and Chan, 1986; Lancet editors, 1990]. In a recently published paper, for example, Narod et al. [1991] found that, although the frequency of genetic conditions (principally Down Syndrome) in childhood leukemia victims was about twenty times as high as in the general population, a hereditary basis was demonstrated in only 2.6% of cases reported to Great Britain's National Registry of Childhood Tumours.

Another hereditary feature which has been studied in leukemics is the HLA or human antigen system. As noted in a previous section, the white blood cells, which are defective in leukemia, are responsible for immunity. The immune response by which organisms differentiate self from nonself and launch reactions against foreign invaders is mediated through glycoproteins (antigens) on the surfaces of white blood cells

[Eisen, 1980]. The genes for these molecules are located in a tightly linked complex on chromosome 6 [Linet, 1985]. The specific array of antigens present in leukemia patients is of interest, because a difference from the general population might indicate increased susceptibility to leukemogenic agents mediated through the presence or absence of a particular antigenic species. Linet [1985] suggests other possible mechanisms including (1) a direct carcinogenic effect of the gene for an elevated antigen, (2) an interaction between the gene or antigen and other genes or gene products, and (3) linkage with oncogenes such that the HLA component is simply a marker for susceptibility to leukemia.

Linet [1985] reports on a number of small (average = 50 cases) case-control studies which, in combination, are difficult to interpret -- the only consistent result being the suggestion of a protective effect conferred by a particular antigen (A9) for ALL.

Two investigators who compared CLL patients to controls, however, found this same antigen to be increased in cases.

In a large study including 1,834 cases consisting of ALL, AML, and CML patients completed in 1987, Bortin et al. found highly significant associations between all types of leukemia studied and the levels of particular HLA antigens (Cw3 and Cw4). The frequency of Aw19 was significantly decreased in AML and CML. These were the only antigens among 32 considered which were significantly related to leukemia. Strengths of this

investigation include its large size and adjustment for multiple significance tests, but the subjects were highly selected (having undergone bone marrow transplants), prevalent cases who had received chemotherapy. Although the authors interpret the data as indicative of the existence of leukemia-susceptibility genes, it is possible that the genes are related more to the ability to survive the disease than contract it.

Chemicals

Although a number of investigations have linked benzene exposure to leukemia occurrence (see Linet [1985] and Austin et al. [1988]), these have mainly been studies of the association between adult leukemia occurrence and previous occupational exposure. Considerable attention has also been given, however, to the possible relationship between childhood leukemia occurrence and parental occupational exposures. Although a better case could perhaps be made for a link between maternal exposures (especially during pregnancy) and childhood cancer, most studies of this subject have concerned paternal exposures -- probably because of the historical predominance of men in the workforce, and particularly in those jobs in which chemical exposure would be likely. Savitz and Chen [1990] have noted that, for father's occupation to be causally linked to childhood cancer, either the child would have to be exposed directly to chemicals brought into the home, or the chemicals would have to alter the father's germ cells.

In 1990, Savitz and Chen found 24 relevant studies to review. All had case-control designs, and most relied on job titles recorded on birth certificates or reported at interview for exposure assessment. Many job titles were found to be associated with childhood cancer occurrence, but there were inconsistencies in the data, which Savitz and Chen thought might have derived from inconsistency of design (e.g., some studies concentrated on particular cancer types, subtypes, or age groups, while others considered childhood cancer or childhood leukemia in the aggregate).

The reviewers seemed to be of the opinion that, because of weaknesses in the study designs, particularly with respect to exposure assessment, the research thus far completed was useful mainly as a screening mechanism -- to focus future investigations on potentially fruitful avenues of research. Savitz and Chen recommended that further attention be paid to paternal paint exposure and paternal hydrocarbon exposure because of the relatively strong and reproducible associations found between jobs that would afford these exposures and childhood leukemia.

From his review, Doll [1989] identified chemotherapy (used to treat cancer and to facilitate organ transplantation) as the only other established chemical risk factor for childhood cancer. He added, however, that, because such treatment causes only AML, its contribution to the total number of childhood leukemias is "trivial."

Tobacco

Little attention has been paid to the possible connection between tobacco use and even adult leukemia occurrence. From a review of the relevant research, Linet [1985], citing relative risks in the range of 1.5 from several large prospective studies, concluded that there is no evidence of a strong association between the leukemias and tobacco use. Austin and Cole [1986], who have also reviewed the data, stated that the Surgeon General's report of 1982 failed to list leukemia as a tobacco-related cancer because studies have not revealed that the risk of leukemia increases with increasing amount smoked (dose-response relationship) -- a trend which is generally sought as evidence of a causal, as opposed to indirect, association.

Another obstacle to acceptance of smoking as a leukemogenic exposure is the failure of studies to find a statistically significant difference in risk between smokers and nonsmokers -- leaving open the possibility that the association might be due to chance. As Wald [1988] has pointed out, though, few studies have been large enough to detect even a 100% increase in leukemia risk.

Results of three case-control studies (see Austin and Cole, [1986]; [Severson, 1987]) indicate that the myelocytic-leukemia risk of smokers may be slightly increased over that of nonsmokers. Each study tested many etiologic hypotheses, however, and each either lacked strong evidence of a link (e.g., no dose-response trend) or suffered from a potentially serious

threat to validity such as small study size or low subject response.

In a case-control study [Schwartzbaum et al., 1991] of childhood cancer in which victims of Rhabdosarcoma were used as controls for leukemia victims, maternal smoking at any time during pregnancy (as determined via maternal interview) was positively (O.R. = 1.5) but not statistically significantly related to ANLL risk in an analysis in which several potentially confounding factors were controlled. The odds ratio relating ALL risk to case-control status was 0.7. These results are difficult to evaluate considering (1) the low participation rate (65.8 percent for all childhood cancer types combined), (2) the potential for smoking mothers to have described themselves as nonsmokers, and (3) the use of victims of another childhood cancer as controls. The similarity between Schwartzbaum et al.'s findings and those of investigations of the link between adult leukemia and smoking (i.e., the magnitude of the odds ratio and the restriction of the association to ANLL cases) is noteworthy, however.

60 Hz Electromagnetic Fields

60 Hz EMF is generated by appliances which use electricity and by all electrical power lines. Common outside sources of 60 Hz EMF are large electrical distribution lines also known as high tension lines which are often seen suspended from large metal or wooden towers. Other outside sources are the power distribution

lines visible on utility poles which are seen on city streets and in most neighborhoods.

Household sources of 60 Hz EMF include kitchen appliances, radios, televisions, electric blankets, hair driers, etc. Less obvious in-home sources include electrical wiring, fuse and circuit breaker boxes and areas in the home near the electrical service drop, the power line which connects the home to the utility pole.

In the work environment significant levels of 60 Hz EMF can be measured near electronically operated machinery, particularly the types often used in high technology industries which manufacture electrical circuitry. Video display terminals are also a common source of 60 Hz EMF at work and increasingly in the home environment as well.

High tension or transmission lines are designed to carry large amounts of electricity over large distances. Before power from these lines can be delivered to homes and industries the voltage must be stepped down to levels suitable for home distribution. This is accomplished at local electrical substations which modify the current and send it along the large primary distribution lines commonly seen on neighborhood utility poles.

As distribution lines travel along a utility network, current is taken from the primary distribution line at certain intervals and passed through the familiar canister shaped transformers commonly seen mounted near the top of utility poles.

These transformers modify the energy flow once more to a form suitable for distribution to secondary power lines which connect directly to residences and industrial structures.

In order to determine the health effects of exposure to 60 Hz EMF it is necessary to identify the levels of exposure a subject is likely to encounter in their environment. Exposure to 60 Hz EMF in a home can be determined by direct measurement of magnetic fields within and around the home environment. Conducting accurate measurements requires significant technical skill, specialized equipment, and open access to the residences in question making this procedure both inconvenient and expensive.

For this reason, Wertheimer and Leeper [1979] developed what they consider to be a surrogate indicator for the prediction of occupants' exposure to 60 Hz EMF which requires much less time to conduct and is less invasive to the home resident. They developed a coding scheme which considers the positions of nearby power lines and transformers and their relative distance from the home and categorizes the likely levels of 60 Hz EMF in the home into four exposure categories. They determined that their prediction method correlated with actual magnetic field measurements taken within homes which had been coded according to their method. Measurements of the electric field did not correlate with their coding scheme.

Scientists had suspected that the magnetic field component plays the primary role in health effects attributable to 60 Hz

EMF. Electric fields can be easily blocked by solid objects and as a result human exposure to them is usually limited. Magnetic fields can penetrate solid objects quite readily. Persons proximal to active power supply hardware would not be shielded from the presence of magnetic fields by walls or other solid objects. Electric fields on the other hand are dramatically weakened by shielding. With regard to 60 Hz EMF then, predominant exposure would result from magnetic rather than electric fields.

In their study, Wertheimer and Leeper classified 344 cancer cases and their controls as having resided in one of four categories of magnetic field exposure during the etiologic period or the time period the illness was likely to be developing prior to its diagnosis (EPA 3-4). Their results indicated that among cases, a significantly greater proportion of persons lived in homes determined to have wire patterns depicting higher magnetic field exposure or high current configuration (HCC) than those coded as likely to have lower exposure or low current configuration (LCC). This effect was stronger for cases and controls who resided at the same address for the entire study period.

In order to address the fact that some subjects had changed address during the etiologic period, Wertheimer and Leeper assessed persons who had moved by considering their birth residence and death residence separately in relation to the control population. For subjects with the same birth and death

address, 44% of the cases had lived in a home considered HCC as compared to 20.3% of corresponding controls. For subjects who had moved, 32.5% of the cases had lived in an HCC home at the time of their birth vs. 20.1% of controls and 37% of the cases had lived in an HCC home just prior to their death vs. 24.0% of controls. These differences are statistically significant ($p < .05$) with those who had only one residence showing the most significance.

Odds ratios were not calculated by Wertheimer and Leeper but subsequent calculations indicate that for subjects with a single address the odds ratio was 3.09 (1.68, 5.71). For those who moved and only the birth residence was considered, the odds ratio was 1.91 (1.09, 3.34). For those who moved and only their death residence was considered, the odds ratio was 1.86 (1.19, 2.91). Urban-suburban differences, socioeconomic class, family cancer history, and gender were considered in the analysis but did not affect significance.

Criticisms focused on speculation that larger power lines were most likely to be found on larger streets their wire codes could thus be surrogate measures for environmental exposures resulting from elevated automobile emissions. Wertheimer and Leeper's subsequent analyses found that although cases tended to live closer to high traffic routes, controlling for traffic patterns did not affect significance.

Wertheimer and Leeper were also criticized for not blinding their researchers to the disease status of residents of homes

they were coding. To address this concern, a blinded study was conducted among a subgroup of persons within the original study's geographic area. Agreement between blinded and unblinded researchers regarding wire codes in the supplemental work was over 90 percent.

In 1980 Fulton et al. attempted to re-create Wertheimer & Leeper's work with 119 leukemia cases diagnosed at a Rhode Island hospital and 238 controls. This population yielded 198 case and 225 control addresses which were measured for exposure to 60 Hz EMF.

Control address exposure values were compared to those of cases for all cancers and by specific type, by stable address, by age at onset of disease, and by socioeconomic status. Although no overall trends were identified, the largest differences between cases and controls were seen in the stable residence population. Frequency distributions were nearly equal for all categories and comparisons of cases and controls.

Fulton attempted to calculate a summary exposure value for study subject residences using an exposure assessment technique which differed from Wertheimer and Leeper's coding scheme. He identified the different types of power lines feeding each home and assigned exposure weights to the different wire types based on field strength values identified in Wertheimer and Leeper's work. He then combined this information with data regarding the distance the wires were from the home to develop a summary exposure value.

In addition to differences in exposure assessment methods, Fulton did not obtain complete residential histories for the control population using only birth residences information in his analysis. As a result of these methodological differences, it is difficult to directly compare Fulton's study outcomes to those of Wertheimer and Leeper.

Tomenius et al. (1982) utilized historical data from the Swedish Cancer Registry in an attempt to reproduce Wertheimer and Leeper's work. They identified 660 malignant and 56 benign tumor diagnoses reported to the registry between 1958-1973 as their case population. Controls were matched on age, sex, and church district of birth and were selected from parish birth registration records.

60 Hz EMF exposure values were estimated utilizing methods similar to those of Fulton (1980). Magnetic field measurements were taken at entrances to single family homes and outside doors to apartment houses. His analysis considered an assessment of the effects of proximity to a variety of electrical constructs including electric railroads, substations, transformers, and subways.

There was a significant excess risk of cancer in case dwellings within 150 meters of 200 Kv wires, O.R. = 2.1 ($p \leq .05$), and a significant excess risk of cancer in dwellings near electrical constructs when all types of constructs were considered together, O.R. = 1.3 ($p \leq .05$). The distributions were also stratified by residential distance from electrical

structures but the risk tended to increase with increasing distance from the structures, a result opposite to what might be anticipated. This stratification resulted in small numbers for some strata and thus unstable statistics for this portion of the analysis.

Results further suggested that the excess risk of residing near 200 Kv lines was seen when data from both birth and diagnosis dwellings was examined. Analyses further indicated that for persons who always lived at the same dwelling, and lived in close proximity to 200 Kv lines, the odds ratio was 3.97 (1.94,8.24) suggesting that when length of residence increased in the vicinity of 200 Kv high tension lines the risk ratio tended to sharpen and increase.

Tomenius also found that a relationship between measured magnetic fields and illness persisted regardless of whether electrical constructions existed near the home. This means that electrical constructions near a home were probably not contributing to magnetic field levels and would thus not be a good surrogate indicator of magnetic field strength.

Savitz et al.(1988) conducted a case-control study of childhood cancer and 60 Hz EMF in the Denver, Colorado area. His study population consisted of all cases of cancer among children aged 14 years or younger residing in the Denver area and diagnosed between 1976 and 1983. Controls were selected by random digit dialing and matched on age, sex, and telephone exchange area. Controls were restricted to children who lived at

their residence at the time of diagnosis of the matched case.

Savitz collected information at interview from study subject parents regarding potential risk factors for childhood cancer. He collected extensive electric and magnetic field measurements at residences occupied by study subjects prior to case diagnosis. Field strengths were measured near the front door, in the child's bedroom, in the parents' bedroom, and any room reported occupied by the child one or more hours per day. Measurements were made both under low power (minimal power use in the home) and high power (lights and appliances in use) circumstances. He used the arithmetic mean of field measurement values as exposure levels for analysis.

Savitz also observed wiring configurations outside homes and used methods based on those developed by Wertheimer and Leeper to establish wiring codes for each of the study subject residences. Thirty six percent of case residences were measured for field values while 75% of the control residences were examined. Wire codes were generated for about 90% of both case and control residences however.

Magnetic field measurements were divided into four exposure categories. The risk of all cancers when low power field values were considered was slightly elevated to a non-statistically significant degree. Odds ratios showed a positive trend across categories ranging from 1.28 (0.67,2.42) to 1.49 (0.62,3.60) for persons residing in lowest to highest magnetic field strength homes respectively. Under high power conditions the risk of

cancer from magnetic fields was weak and did not demonstrate a consistent trend with odds ratios close to unity. Under all circumstances, when electric field strengths were considered no increased risk of cancer was identified.

Magnetic field exposure values were dichotomized into two categories,
$\geq$ 2.0 Mg and < 2.0 Mg, and the data was examined for more specific cancer type. Nonsignificantly elevated risks were observed for leukemia, lymphoma, brain, and soft tissue cancers under low power conditions. The highest odds ratio, 3.26, (0.88,12.07) was seen for soft tissue sarcomas and magnetic fields and for lymphoma, 2.17,(0.46,10.31).

High power analyses demonstrated somewhat reduced risks in most cases. For leukemia, an elevated odds ratio of 1.93,(0.67,5.56) for $\geq$ 2 mG magnetic fields at high power was identified. An odds ratio of 0.75,(0.29,1.91) was seen for electric fields at high power.

It might have been expected that high power conditions would have produced higher risk estimates for magnetic field levels. It is likely however that high power circumstances reflect the most transient magnetic fields and the most varied exposures. Circumstances of low power exposure would likely represent a more stable exposure level most significant to disease etiology. This effect could also be the result of a poor response rate among cases, 36% of case residences were measured for electric and magnetic fields, 74.5% of residences of corresponding controls

were measured, thus a poor estimate of typical magnetic field strengths in case homes.

The fundamental conclusion from Savitz's field measurement work was that a modest increase in risk occurs for some cancers in homes where low power magnetic fields were greater than or equal to 2mG. These relationships went largely unchanged when estimates were adjusted for potential confounders. Adjusted odds ratios for leukemias varied from 1.8 to 2.4 while the unadjusted value was 1.9.

When Savitz examined wire codes of subject residences his results indicated that for cancer at all sites the highest risk was for very high wire code homes, the non-significant odds ratio equalling 2.2. For homes which were occupied two years before diagnosis the statistically significant odds ratio was 5.22. Tests for trend were statistically significant providing evidence of a dose-response relationship.

For the site-specific cancers which he examined (leukemia, lymphoma, brain, soft tissue, and other cancers) odds ratios when considering a dichotomous wire code analysis were elevated for each cancer site except lymphoma. The odds ratio for brain cancer was the highest at 2.04,(1.11,3.76). For leukemia the odds ratio was 1.28,(0.90.2.63). When only the most extreme codes were compared (lowest current configuration to highest), for leukemia the risk was 2.75 and for brain 1.94. There were too few subjects in these exposure categories to consider statistical significance. Elevated odds ratios were also found

when wire codes for the residence at diagnosis were examined for all sites combined. Analysis for confounders revealed no significant change in odds ratios.

Savitz confirmed a positive association between 60 Hz EMF exposure as represented by wire codes and the incidence of leukemia, lymphoma, and brain cancer. A significant dose-response relationship existed based on total cancers and wire code configurations at the time of diagnosis as well as 2 years before. His evidence supports the use of wire configuration codes as surrogates for magnetic field exposure measurements.

If wire configurations are identified as valid surrogate measures for 60 Hz EMF exposure, they would represent a significantly more accurate historical record of the electric and magnetic field environment of the home than field measurements would likely be. Wiring configurations in most neighborhoods are likely to be stable over time while home electric and magnetic field measurements are likely to be in a constant state of flux.

A recent work addressing 60 Hz EMF and cancer was conducted by London et al. (1991). This research focused specifically on exposure to electric and magnetic fields and the risk of childhood leukemia. The authors examined the relationship between electric and magnetic field measurements in homes and wiring configurations, self-reported appliance use, and the incidence of leukemia among children from birth to 10 years of age.

Data used in London's analysis were 24 hour measurements of magnetic fields in the subject child's bedroom obtained for 164 cases and 144 controls, spot measurements of magnetic and electric fields for 140 cases and 144 controls, and wiring configuration coding values for 219 cases and 207 controls.

Researchers determined that they would gather exposure data for the residence in which the study subjects had lived for the longest period of time during the "etiologic" period. The etiologic period was defined as the interval between the last known menstrual period preceding the birth of the case and the date of the case's leukemia diagnosis. Forty three percent of the cases and 34% of the controls had lived in the same home for the entire etiologic period and were included in the study. For more than half the study subjects the measurements covered at least fifty percent of the etiologic period.

Wiring configuration was also examined and used as a predictor of exposure to electric and magnetic fields. This was done for residences not accessible for measurements as well as those that were measured for field levels. Field technicians were trained according to the system of Wertheimer and Leeper as done in previous studies.

Technicians conducted residential measurements to determine domestic electric and magnetic field levels. The protocol attempted to address the effects of variation of field strength by time of day, location within the home, and power use patterns.

Measurements were taken in several locations within the home and outdoors.

The researchers found that in general the field exposures measured in the homes were in agreement with the predicted levels based on the Wertheimer-
Leeper model. Homes predicted to be in lower current categories had the lowest measured values and vice versa for magnetic fields. Cross-category trends were seen for magnetic field measurement parameters.

The leukemia risk was evaluated in relation to wire codes and field measurements. Twenty-four hour magnetic field measurements showed no consistent trend with leukemia although the highest field level demonstrated the highest risk. Spot measurements of low power magnetic fields were not correlated to leukemia risk. The same pattern was seen with electric fields.

When the Wertheimer-Leeper wire codes were analyzed, the lowest level above-ground wire code was combined with the very low underground category to create a single reference group. There was a significant association classification and leukemia risk even after controlling for potential confounders. The association was slightly stronger when the analysis was limited to the residence lived in the longest when compared to an analysis of multiple residences for subjects when moved.

In 1993, Feychting and Ahlbom [1993] released the results of a study which was conducted to test the hypothesis that exposure

to magnetic fields from high voltage power lines increased cancer incidence.

The study was a nested case-control design based on a study population of cases and controls who lived in residences within 300 meters from any of the 220 and 400 ks power lines in Sweden between 1960 and 1985. For children, all types of cancers were considered, for adults the study was limited to brain cancer and leukemia. Cases were identified through the Swedish Cancer Registry and controls were matched to cases based on time of diagnosis, age, sex, parish of residence and the type of power line they lived near.

The authors utilized three different exposure variables. First, spot measurements of magnetic fields were collected in the homes where the study subjects lived at the time of the diagnosis of the case. Four five minute measurement periods were used in each home according to the procedures used by Savitz et al., (1988).

Second, historical field information was calculated for each of the high tension lines using a computer program which considered distance, line configuration, and historical information regarding line loads. Historical load data that were kept by power station managers. Based on these calculations the exposure metric was the annual average calculated magnetic field generated by the line. The calculations considered confirmations of the wires on the towers, load on the line, position of other

nearby lines, location of homes relative to the towers and altitude of the homes and lines.

Third, for a sample of the subjects a personal dosimeter was used to obtain 24 hour measurements. This sample was of 100 subjects always including one child and one adult from the same family.

Data was stratified by age, sex, and year of diagnosis as well as county of residence and type of dwelling. Analyses controlled for socioeconomic status and air pollution from traffic patterns. Analyses of magnetic field strengths considered a variety of category cut off points so that conclusions would not be sensitive to random decision categorical effects.

The results indicated that for historical magnetic fields and cancer in children, the relative risk for leukemia increased over two exposure levels, O.R. = 2.7 (1.0, 6.3) for a historical negative field value of 0.2 microtelsa (uT) which is the equivalent of 2 mG. The test for trend was significant p = 0.02). When the exposure category was redefined with 3mG as the lowest upper bound of the high exposure category the relative risk increased to 3.8 (1.4, 9.3). The test for trend was again significant (p=0.005). For brain tumors among children and for all cancers together there was no increased risk identified.

The analyses for spot measurements and childhood cancer indicated that there was no excess risk for total cancer or for leukemia while there was an elevated but borderline

nonsignificant risk for brain cancer for the intermediate exposure level (1 to 2 mG) O.R. = 2.5 (0.9-6.6) which decreases at exposure over 2mG, O.R. = 1.5 (0.4, 4.9).

When cancer risk was analyzed in relation to residential distance from the power line there was an elevated leukemia risk for those living closer than 50 meters from the line compared to those living more than 100 meters away, O.R. = 2.9 (1.0, 7.3)

For adults certain leukemias showed a slight nonsignificant elevation in risk in the highest magnetic field exposure category R.R. = 1.7 (0.7-3.8). For other categories or exposure levels there were no noticeable risk elevations.

The type of exposure assessment performed in this study assessed magnetic field exposure only in relation to transmission lines. The authors suggest that a model could be developed to estimate magnetic field exposure outside the home relative to sources inside the home as well. They note that although some magnetic field sources are ignored in the exposure assessment, the same sources are ignored in both the cases and controls and should not give rise to spurious associations between magnetic fields and cancer, rather likely dilute associations.

Ionizing Radiation

Leukemia as a late effect of radiation exposure has been recognized since 1944 (a decade after Marie Curie's death from radiation-induced leukemia [Lancet editors, 1984]), when March reported that this disease was nine times more commonly recorded

as a cause of death among radiologists versus other physicians [Linos et al., 1980; Gee, 1987; Linet, 1985; Berry, 1987]. Today, cancer is well recognized as the principal health hazard associated with exposure to small doses of ionizing radiation [Fry, 1981; Rossi, 1980; Rahn et al. 1984; Upton, 1982; Gofman, 1979], most of our understanding of the carcinogenic effects having been gleaned from studies of survivors of the atomic-bomb detonations that ended World War II. Although the A-bomb studies have shown an indisputable link between high-dose ionizing radiation exposure and leukemia [Mole, 1975], data analyses have suggested a much lower cancer rate than expected among cohort members who were in utero at the time of the bomb (ATB) [Sztanyik, 1978; Kato, 1978]. Although Mole [1975] suggested that the apparent insensitivity of those in utero ATB, supports ". . . the thesis that sensitivity to carcinogenesis increases in general with increasing age," this does not appear to be the case for those not in utero ATB, because, also according to Mole [1975]: "Overall leukemia induction in those exposed as children and young people aged 0-19 years was at a slightly larger rate than for adults aged 20+ years when irradiated . . . ."

One possible alternative to the "insensitive fetus" hypothesis as an explanation for the low yield of leukemia fatalities among those irradiated in utero relates to the abnormal condition of the populations of Hiroshima and Nagasaki prior to and following the 1945 bomb blasts, in conjunction with the fact that follow-up of the survivors did not begin until

1950. A number of authors have commented over the years on the extreme stress inflicted on the survivors and the deprived state of the population during the war years. Nussbaum [1985] reminds us that the Japanese were exposed not only to the bomb but also, only a few weeks later, to a typhoon followed by an unusually harsh winter (1945 - 1946). This author claims that, as a result, bomb survivors were without adequate food, shelter, sanitation, and medical services for several years. A point which has often been made in the recently published literature and attributed to Stewart and Kneale [Nussbaum, 1985] concerns the probability that some of those with immune systems weakened by radiation-induced leukemic changes may have died of acute health problems (e.g., infections) without an official diagnosis of leukemia ever having been made. Even without invoking this particular hypothesis, it seems reasonable that the proportion of the population surviving the great deprivation of the time would have been unusually robust as compared to a "normal" population -- the implication being that perhaps some of those destined to develop radiation-induced cancer were lost from the population before follow-up began. Arguing against this idea, though, is (1) the absence of any obvious effect of the circumstances on the general mortality experience of the survivors (i.e., noncancer mortality rates have been normal, as opposed to decreased, when compared to unaffected parts of Japan [Beebe et al., 1978]) and (2) the absence from the population of its very strongest members -- military personnel.

Stewart and Kneale [1970] have reported that noncancer deaths in general increase with dose for survivors exposed to ≥ 50 rad but that the "healthy-survivor" effect is apparent below this exposure level exposure level [Nussbaum, 1985]. These authors attribute the high noncancer morality of the heavily exposed to bone marrow damage evidenced by the high incidence of aplastic anemia.

The low leukemia death rates found among those in utero ATB (20 cancer deaths per million per rad [Mole, 1975]) can be contrasted with the elevated leukemia rates (240 per million per rad [Mole, 1975]) associated with prenatal X-ray exposure in the Oxford Survey of Childhood Cancers (OSCC) [Knox et al. 1987] -- a finding which was at first rejected because of its disagreement with the A-bomb data as well as certain design issues which were resolved in more recently conducted studies of the same phenomenon [Doll, 1989]. Interesting in this regard are cytogenetic data which have suggested that X-rays may be three times as effective as gamma rays at causing certain chromosomal aberrations at low doses [NCRP No. 64, 1980].

APPENDIX II

Peer Review Committee Comments

THE UNIVERSITY OF NORTH CAROLINA
AT
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August 29, 1995

Kevin S. Costas, M.P.H.
Bureau of Environmental Health Assessment
Massachusetts Department of Public Health
150 Tremont Street
Boston, MA 02111

Dear Mr. Costas:

On behalf of my fellow peer review committee members, Drs. Nancy Kim and Edward Stanek, we would like to submit the attached final report. As it states, we were most impressed with the careful work and documentation the report represents. We focused on areas of concern that we believe could be improved, ranging from broad topics to rather minor editorial points (listed at the end).

If you would like clarification or a conference call to discuss our report, please let us know. Thank you for inviting us to offer our comments.

Sincerely,

David A. Savitz, Ph.D.
Professor

cc: Nancy Kim, Ph.D.
Edward Stanek, Ph.D.

Review of Woburn Childhood Leukemia Study
Final Report
August 28, 1995

Review Committee:

Nancy Kim, New York State Department of Health
David A. Savitz (Chair), University of North Carolina School of Public Health
Edward Stanek, University of Massachusetts at Amherst School of Public Health

General Comments

Strengths

The report on the case-control study of childhood leukemia in Woburn provides detailed documentation of a well-designed and carefully conducted study. The basic structure of the study is well-suited to addressing the potential relationship between consumption of contaminated water from Wells G and H in relation to childhood leukemia as well as some other environmental exposures. It does not address whether there is some community-wide exposure that has elevated risk for all Woburn residents since the study is restricted to that community.

The methods used by the authors are current and appropriate. Methods for case and control assessment, and construction of variables are presented in detail. Analyses are developed methodically and also presented in the same level of detail.

Imprecision

The principle limitation in the study is well-recognized by the investigators, namely the limited number of leukemia cases that occurred in this community. The consequences of this limited study size are: a) imprecise estimates of associations between exposures and disease and failure of relative risks that are not extremely large (around 10) to attain conventional levels of statistical significance; b) limited opportunity to examine dose-response gradients; and c) difficulty in making statistical adjustments for confounders.

As a result of the imprecision in risk estimates, there is an unusual degree of latitude on the part of the investigators to make judgments about what is and is not worthy of attention. The conclusions of the study may be too strong in light of this imprecison. The patterns of association between potential consumption of contaminated water and risk of leukemia are worthy of note, but persuasiveness of those results may be less than the report suggests. In particular, the Executive Summary should be reviewed and modified so that those who read only the summary are aware of the limitations that apply to the study's conclusion

Water Exposure Assessment

The other key challenge is inherent in studies of environmental causes of cancer, namely difficulty in identifying the historical exposures of concern. A strength of this study is the availability of an exposure model

that generates credible estimates of the likelihood of having consumed contaminated water from wells G and H, a function of geographic location and calendar time period. The report does not include a discussion of the limitations and uncertainties in the exposure model. Moreover, how these limitations or unknowns could affect or bias the findings of this study is not discussed, which would be useful.

The description of the trichotomous exposure score needs to be expanded with further justification added. The boundaries for the two non-zero exposure groups were selected so that the two groups would have equal numbers of cases and controls, and not on the basis of differences in exposure scores. No information is given as to the average or median exposure score for each of the different exposure time periods and the maximum exposure score is not given for the different exposure time periods. The report emphasizes the dose-response relationship, but the information given is not sufficient to show that the two non-zero dose groups really have meaningfully different exposures. The data inconsistencies in exposure categories between Tables 6 and 10 need to be addressed.

Some additional refinements in the water exposure assessment would be useful. The effort to consider consumption of tap water is useful, but it would be more informative if examined as part of a dose-response gradient. The lowest exposure group might be no contaminated water, a middle group that received but probably did not drink contaminated water, and a third group that received and drank contaminated water.

It is not clear whether any of the cases diagnosed after 1979 had the potential for exposure to Wells G and H during pregnancy given when use of the wells ceased. Not enough information is presented to evaluate this. Some comment regarding the consequences of the limited calendar time of potential exposure for cumulative exposure scores is warranted. It seems that since exposure is not possible after 1979, calculation of a cumulative exposure measure up to 1987 will dilute the exposure levels, for some subjects relative to those who could have been exposed throughout their pregnancies.

The use of different exposure cut points is potentially problematic in interpreting the results in Table 11. If exposure is expressed per unit time, the cut points and scales may be more comparable.

A useful calculation might be to assess what proportion of cases are potentially attributable to consumption of contaminated water before and during pregnancy. Does this exposure potentially account for a sizable fraction of the excess risk observed in Woburn?

Control Selection

A key concern in this study, as in all case-control studies, is the suitability of the control group as a representative sampling from the study base that generated the cases. Some additional tabulations and discussion might help in judging the adequacy of controls. The overall strategy of sampling randomly from the entire community of Woburn is appropriate. School records provide a potentially useful sampling frame for generating the desired sample, but in spite of the detailed explanation of procedures, it is not clear whether the sampling frame for controls fully identifies children who would have been eligible for the study had it been done concurrently. That is, if the risk set had been fully enumerated at the time each case developed, how would that risk set compare to those who actually constituted the sampling frame for controls. Potential concerns include selection of controls for pre-school age children, children who attended private schools, and loss of

potential controls due to having moved after the relevant diagnosis date. Beyond the question of whether the targeted controls are appropriate is the question of whether non-response may have distorted the patterns.

Because the water exposure model generates estimates based on both location of residence and time period of occupancy, it is important to be certain that the sample of controls is unbiased with respect to those factors. A more direct discussion of the potential for bias in control selection is warranted, even though it is not at all obvious that such bias has occurred. Along with random error, it is a key source of uncertainty in judging the validity of the study's results.

Confounding

The two strategies for controlling confounding were to use a composite score or include all pre-selected covariates in a logistic regression model. An alternative not considered was selective inclusion of only the subset of individual variables that generated a change in the coefficient for the variable of interest. In fact, there was little effort to identify the potential risk factors that actually appeared to be associated with childhood leukemia in these data (e.g., dental x-rays) as a restricted set of potential confounders to be controlled in the analysis. It appears that there was little confounding overall and it is quite possible that adjustment for one or two covariates would have been sufficient to control confounding without much loss in precision relative to the unadjusted results.

Miscellaneous

Results of the study are comprehensively presented, including the needed descriptive tabulations and unadjusted measures of association. Examination of risk for specific time windows is a very useful strategy. Although the imprecision of risk estimates is known to be a major source of uncertainty, the repeated notation of which measures of association are and are not statistically significant is unnecessary.

The other potential risk factors were collected in the usual manner for such studies, including job titles of the mother and father, health behaviors of the parents such as tobacco and alcohol use, and medical history. A special effort was devoted to examining proximity of residences to power lines in order to address wire configuration code of the home as a potential risk factor. Again, the overall design and array of risk factors considered are appropriate to the situation of concern.

Some specific observations were not given as much attention or comment as they might deserve. There were relatively large (although non-statistically significant) differences in risk observed in Table 13 when accounting for types of leukemia. This table appears to suggest a very large effect for AML/CML cases on the risk (since the risk is elevated from 2.8 to 5.7 based solely on 4 more cases), which should be noted and interpreted in the text.

A more extensive effort to compile the literature specific to environmental exposures and childhood leukemia would be helpful in providing the context for these results. Although studies are cited in a general way, the evidence specifically on exposure to organic solvents during pregnancy and subsequent risk of leukemia in the offspring should be scrutinized, for example. Other peripherally relevant studies of these agents in relation to

adult leukemias, birth defects and other adverse pregnancy outcomes, or health effects in general should be considered. A firm judgment of whether contaminated water caused the increased risk of leukemia is not possible but a better assessment of that possibility could be made based on more details of other relevant studies.

David Savitz - Specific Comments

A minor concern throughout the report is the need for careful technical editing to correct spelling, grammar, and punctuation errors. It should be noted that "data" is plural.

Executive Summary

Page

2 The general methods of control selection should be described.

2 Selection of results for presentation solely on the basis of statistical significance rather than the interest or importance of the question is not a good strategy.

3 Results for dose-response analysis should include estimates of relative risk, not just p-values for trend.

3 The breastfeeding results should be tempered given contradictory evidence in other studies.

Introduction

4 Paragraph 1: Is the absence of an unusual geographic pattern in childhood leukemia mortality discrepant with a geographic pattern to the exposure?

24 In the sections on solvents, tobacco, etc., there needs to be a much clearer separation of evidence related to childhood leukemia versus adult leukemia. Current wisdom would suggest that the adult leukemia studies may have little relevance to risk factors for childhood leukemia.

27 Some literature citations are needed in the first paragraph.

Methods

32 Matching in case-control studies does not control confounding but creates balanced groups that, when properly analyzed, make it easier to control confounding.

42 Bottom: Did cases have a similar restriction to that described for controls with regard to when they had to have attended school?

59 Middle paragraph: Trimesters are 13 rather than 15 weeks.

67 Paragraph 2, last sentence: Should say "lowest exposure group" not tertile.

69 In discussion confounding, it should be acknowledged that we actually know very little about determinants of childhood leukemia and that the list includes many variables of potential interest which may well not be risk factors at all.

70 11 lines from bottom: How did you define "significantly affect" in quantitative terms?

Results

72 Bottom: What was the final effective control response proportion?

73 Distinguish between ineligible (who should not be counted in calculating response proportion) and refusals (who should be counted).

74 In selecting parents, it is not clear whether distinctions were made for biological versus adoptive parents, and whether fathers and mothers had to be currently married for the father to be interviewed. This should be clarified in the Methods section.

75 First full sentence: Something is wrong with sentence.

76 8-10 lines from bottom: This is confusing and not quite correct in describing what an odds ratio is. It is the odds of exposure among cases versus controls; nonexposed cases are the denominator of the odds of exposure among cases and similarly for the controls.

83 Line 6: Here and elsewhere that statistical tests are performed, it would be preferable to provide the actual p-value rather than to note only whether or not it was significant.

86 and later: Selection of statistically significant results for presentation is not a good strategy. Given the need to be selective since so many odds ratios were generated, those that are based on questions of interest and where prevalence is adequate to study would be the most important, regardless of whether an association was found. Also, there is no clear logic to the sequence of the presentation, making it hard to follow the flow of information. Environmental exposures might be grouped, for example.

91, 92: Presentation of a summary of results (not just in the Appendix) would be useful.

92 10 lines from bottom: Which direction was the age association -- higher risk with older or younger maternal age?

93 Bottom: Put employment information with occupation results.

94 Paragraph 3: Why doesn't 6 exposed controls and 0 exposed cases generate an odds ratio of 0?

95 Bottom: The low prevalence of breastfeeding (presumably for any period of time) is so notable that it calls into question the results. This might be mentioned in the Discussion.

99-102 The EMF results are made far more convoluted and confusing than they need to be. You could start by creating variables for specific times such as "wire code at birth" or "wire code at diagnosis" to analyze and avoid pools of homes without regard to time of occupancy. Given measurement data, there are three discrete groups: very high current configuration, ordinary high current

configuration, and everything else. You could group the lowest categories together and present the results for the other contrasts (OHCC vs. low, VHCC vs. low). It is not necessary (and is somewhat confusing) to present so many others.

Discussion

113 The contradictory literature on breastfeeding and childhood cancer should be cited.

114 4 lines from bottom: Underground wiring is not assumed to have zero exposure but rather to be in the lowest group.

116 Bottom: It is not clear why the observed distribution of wire code categories suggests that "Woburn's neighborhoods are older..."

Ed Stanek - Specific Comments

Executive Summary

p2, line -11. Wording is awkward. "as univariates" means what?

P3, paragraph 3. Results summary here is not clear. Suggest use of " 3 levels of exposure" in lieu of "trichotomous" or explain more clearly.

P3, line -4. I suggest rewording something like: "Our findings are consistent with the hypothesis that low level multi chemical exposure, which may have occurred as a result of consuming water form Wells G and H has a hematologic effect on the human fetus, ultimately resulting in leukemia."

P4, line 1. Begin with "Other studies ..."

P4, line 4. Suggest rewording: "This study concludes that the risk of developing childhood ..."

P4, line -7. Suggest rewording: "Further study by other researchers may be warranted to examine ..."

Report

p3. Line 3. Add comma: "Woburn residents, having..."

P3, Line 4 Add comma: "... may have caused, became ..."

P13. Pagination.

P17, lines 1-6. This discussion is not clear to me. Perhaps it can be restated.
P19, line -11 to -8 I don't see how the difference in the spike will result in the conclusion you attribute to it.

P28, lines 3-5. The sentence is awkward.

P35, line 4. Include reference for "1981 study"

P55, top paragraph. The evaluation of the water model indicates that there will be a substantial miss-classification of exposure measures for individual subjects based on the test data. Such misclassification will bias risk estimates down. Does this deserve some discussion?

P56, line -12,-11 The sentence appears incomplete.

P66, line -8. I feel you should expand on your description of the model. You use a proportional hazards model to fit the conditional logistic regression model to case/control status. The report never seems to state that clearly, nor to reference the connection between the analysis. This should be amplified. There is some discussion of this on page 71. However, you never write out a model equation, or reference the method (other than a general reference to the SAS program).

P67, line 5. "All data were..."

P68, line-6. Use of Tukey's suggestion for collapsing covariates may be reasonable. Additional information that would have been valuable is an evaluation of the sensitivity of the choice of coefficients for the collapsing, since the method seems somewhat at hoc.

P74, line 9. Figure 1 is unclear. It would help to identify the interval for case detection that you have used when plotting cases.

p76, line 6. I don't find the ranges reported in Table 7 of much value. Why not report medians, quartiles, or other descriptive statistics.

P76, line 14. Is this a line that should be edited out? Where does 51.07 come from?

P78, line -12. "confounders that were considered, ..."

P80, line -11. I am missing the rational for the trimester analysis, other than being another interesting thing to look at. Do you have any rational for expecting a difference in one direction or another? It seems somewhat like a fishing expedition, and has principally descriptive value. What conclusions do you make from your analysis results?

P83, lines 5-9. Did you do any analysis to evaluate the sensitivity to your dose classifications?

P84, lines 14,15. The degree of confidence is the same for both confidence intervals. One is just narrower, so the estimate is probably better. This should be reworded.

P85, line 3-4. "As a univariate,..." Reword this. Also, some mention of , or reference to recall bias here may be warranted (I know you address it later).

P85, lines -6 to -4. None of the results are significant. The ALL results are weaker. You seem to be "beating the drum" here.

P96, line -10. This is one of the few results statistically significant, but is under billed. More discussion is warranted. Do you consider it a "chance" finding?

P110, line 12. Reword this sentence. One estimate is a better estimate than the other since it appears to be more precise.

P113, lines 15-19. More discussion (and possible analysis) of how breast feeding fits into the picture for incidence with the other variables is warranted.

P117. Line -9. "association [of what?] with each ...."

P117, line -7. Is Table 13 the right table to reference?

P122, 2nd paragraph. I don't understand why the discussion brings in "sensitive individuals", or how this adds to interpretation of the results.

P125, lines -12 to -8. I think this explanation is not very plausible. Are subjects equally aware of other risk factors? Wouldn't they have to be to bias the results negatively?

P126, line 8. "...leukemia was greater for ..." I think this should be toned down.

P126, line -1. "...is necessary to definitively ..." I think this should be toned down.

P128, line 6. Should some mention be given to the possible explanation that the elevated rates were simply the result of natural variation?

Nancy Kim - Specific Comments

P 12, Paragraph 2. "1,1,1-trichloroethylene" spelled incorrectly; it should probably be 1,1,1-trichloroethane.

P 25, Sentence 1. "studies,are" missing a space.

P 45, Paragraph 1. "Duplicate questions asked of fathers if parents were interviewed apart to validate or complete missing information from one or the other parent." Something is wrong with the sentence structure. Should "were" be inserted after "questions"?

P 49, Line 1. "pi;e" should be replace with "pipe".

P 49, Paragraph 1. "It was assumed that leakage flow was uniformly distributed through the system." Given this assumption, what is its basis and could any errors be introduced?

P 49, Paragraph 1. "...water consumption was estimated by applying the value

of 370 gallons per day to each residential..." What is the basis for this assumption?

P 49, Paragraph 2. "The amount of residential and...." Should the word "assumed" be inserted before amount? Based on the earlier discussion, this would seem necessary.

P 52, Paragraph 1. "Root mean square head difference was 6.2 feet." Can you give the reader a qualitative idea as to the importance of the error level and root mean square head difference?

P 54, Paragraph 1. "Overall, 72 percent of the field test predictions were within 0.2 of the observed values." Should the value 0.2 have units?

P 56, Paragraph 3. "The monthly exposure index value for each user demand area used in the calculation of the exposure index values for each individual study subject." Something is wrong with the sentence structure.

P 89, Paragraph 1. "Once again, odds ratios demonstrate no association between occupational categories most frequently reported and childhood leukemia incidence." I don't disagree with this statement although I was troubled in reading this section by what appears to be inconsistencies as to which comparisons are pointed out and discussed and which were ignored. The selection is not statistical significance, but what is it? For example, is it an odds ratio of 1.5 and that is why the fathers in the durable goods manufacturing industry were discussed and why mothers in the clerical category weren't, e.g. 1.98 (0.63, 4.72) and 1.40 (0.50, 3.97)?

P 99, Paragraph 3. "Forty-six of the seventy-three study subject addresses (63%) were classified as OLCC." I may have missed it, but I couldn't find the definition of OLCC, VHCC, etc.

P 105, Paragraph 2. "...with the first study and 4) Electric power..." Lower the case of "e" to "electric."

P 108, Sentence 3. "Although confidence intervals are extremely wide for odds ratios of individual pregnancy strata, a test for trend is significant suggesting the existence of a dose-response relationship between Wells G and H water and childhood leukemia for exposure during pregnancy." I am uncomfortable with this and some of the other statements which focus on the importance of the dose-response relationship, given the description of the exposure data. However, if additional data clearly show a difference in exposure between the two non-zero dose groups, my concerns will be diminished.

P 109, Sentence 1. "The tap water question must be interpreted with caution however as respondents were likely to be aware of the community's concerns regarding ill health and exposure to contaminated public water." This is a reasonable qualifier, but these types of qualifications are not carried over into the executive summary.

P 116, Paragraph 2. "Odds ratios resulting from the relationship between wire code configuration and childhood leukemia do not support a positive association between wire code and leukemia incidence." As mentioned previously, the criteria for stating when odds ratios, not statistically significant, are considered a positive association and when they are not need

to be defined.

P 116, Paragraph 2. "...approximately 80% of the residences were int the two..." typo, delete the "t" at the end of the word "in".

P 121, Sentence 1. "...over which time periods, it would therefore be impossible..." change to "...over which time periods. It would, therefore, be impossible."

P 121, Paragraph 3. "The environmental protection Agency's" change to "The Environmental Protection Agency's"

P 122, Paragraph 2. Add a comma after fetus in "...effects of TCE on a developing fetus however, the plausibility...". In addition, the results from animal reproductive studies, either positive or negative, should be mentioned.

P 123, Paragraph 1. "Alternatively, fetal susceptibility to potential drinking water contaminants could reflect high dose exposure. Regular maternal ingestion could lead to sustained blood levels or even concentrations in fetal tissues." This sentence should be rewritten so the meaning is clear. In fact, the statement may not be necessary, given the sentence that follows.

P 126, Paragraph 1. "...showed a significantly positive relationship to the amount of water mothers consumed." Would the following statement be more accurate? ""showed a significantly positive relationship to the contaminant levels in the water delivered to the households."

P 128, Paragraph 1. "Ongoing cancer incidence surveillance will provide information to suggest whether control of environmental hazards in Woburn has coincided with a more predictable rate of childhood leukemia incidence." Isn't in more accurate to refer to the control of specific contaminants in drinking water rather than environmental hazards?

Table 10 Some of the data in this table are not consistent with the data in Table 6. The number of cases in the never exposure category for the exposure time period from 2 years before conception diagnosis is 4 in Table 6; however, the number of cases with the 0.00 exposure score in Table 10 for the same exposure period is 3. A similar problem occurs with the exposure time period "during pregnancy". In addition, the third exposure category for most of the time periods has a "<" sign before it; the sign should be ">". In Table 11, some of the signs are omitted and need to be added.

Table 15 and 16 Combine "Transportation" and "Communications and Other Public Utilities" into one category "Transportation, Communications and other Public Utilities."

Responses to Peer Review Comments

This section addresses all peer review comments received concerning the Woburn Childhood Leukemia Follow-up Study. The Peer Review Panel members were David Savitz, Ph.D., (Chair), University of North Carolina School of Public Health, Nancy Kim, Ph.D., New York State Department of Health, and Edward Stanek, Ph.D., University of Massachusetts at Amherst School of Public Health.

The study was sent to the peer review committee on July 12, 1995 and a final report was received from the committee on August 28, 1995. Grammatical changes suggested by the peer reviewers are not listed herein but were largely incorporated. A copy of the actual peer review committee report appears at the end of this response section.

* Page 1, paragraph 4 of the comments states, "The patterns of association between potential consumption of contaminated water and risk of leukemia are worthy of note, but persuasiveness of those results may be less than the report suggests. In particular, the Executive Summary should be reviewed and modified so that those who read only the summary are aware of the limitations that apply to the study's conclusion."

The narrative of this report was modified to more closely reflect the context within which the results should be viewed. As an example, the following paragraph was added to the Discussion and Conclusions section of the Executive Summary to better illustrate the study's limitations: "The small number of study subjects lead to imprecise estimates of leukemia risk. Because of the imprecision of the risk estimates, the exact nature and magnitude of an association between exposure to water from Wells G and H and risk of childhood leukemia cannot be stated. However, results consistently were in the direction of an association and support the hypothesis that childhood leukemia in this population may be related to mother's exposure to contaminated water from Wells G and H during pregnancy."

* Page 2, line 3 of the comments requests that more information be provided regarding the limitations and uncertainties of the exposure model. It states, "A strength of this study is the availability of an exposure model that generates credible estimates of the likelihood of having consumed contaminated water from Wells G and H, a function of geographic location and calendar time period. The report does not include a discussion of the limitations and uncertainties in the exposure model. Moreover, how these limitations or unknowns could affect or bias the findings of this study is not discussed, which would be useful."

In order to assess the potential uncertainty of the model, validation studies of the Murphy Model were conducted by Dr. Murphy during the model's development. Known concentrations of fluoride were added to the Woburn water system. The model was used to predict concentrations of fluoride that would be measured from samples taken downstream after normal water system mixing had occurred.

The model was used to predict three overall zones of water exposure and their boundaries within Woburn, a zone of no possible contaminated water exposure, a zone of definite maximum contaminated water exposure, and a mixed zone whose water contamination level likely fluctuated over time.

The results of the validation study indicated that the model had the potential to error in the placement of the boundaries for the all, none, or mixed water exposure areas by a factor equivalent to a maximum of one node in either direction at the borders of the major exposure zones. Within the mixed exposure zone, the model predicted the actual mixture concentrations with an average error of 10%. Dr. Murphy noted that nearly 70% of the validation measurements were within 20% of the concentrations predicted by the Murphy Model.

Given the predicted "one node in either direction" maximum error, and the maximum potential 10% error for the contaminated water

concentrations in the mixing zone, we performed a sensitivity analysis using our exposure estimates. We created an artificial circumstance for which the model would maximize the errors in exposure estimates so that differences in exposure estimates between cases and their matched controls would be greatest. Resultant exposure values illustrated the widest range of risk estimates possible based on the water model. Outcomes predictably ranged from risk values suggesting a protective effect of contaminated water exposure to an extreme risk of adverse outcome from exposure. This is expected based on deliberate maximization of the potential errors inherent in the calculation of error in each node, i.e. maximum error for each node exposure estimate. These extremes in the model's predictions can only result when the maximum error occurs for each node during every month the model predicts exposure values.

Murphy Model peer reviewers have determined based on Dr. Murphy's validation testing that the water model predicts contaminant concentrations consistently within an acceptable range of error. It would be highly improbable then that the model when applied to our data would have consistently demonstrated errors of greater magnitude than demonstrated in the validation study.

The extreme error we demonstrated in our sensitivity analysis is virtually impossible under any circumstance. For this extreme error to occur the model must error for each node exposure

estimate for each study subject's month of residence to the greatest extent possible. This must occur in a consistent direction for cases and consistently opposite direction for controls in order to maximize the difference in exposure estimates between cases and controls and therefore have the most effect on risk estimates.

If we assume the Murphy Model to be valid, as indicated by the Murphy Model peer review committee, then the amount of error for each node's predicted exposure value would be within a known range relative to the actual exposure value. Any potential bias in exposure value estimates would therefore be the result of predictably limited error. This error would be distributed among all nodes and would not occur more often than expected in any particular node thus giving reasonable assurance for unbiased results.

* Page 2, first full paragraph of the peer review comments asks for further description and justification of the trichotomous exposure score. They state "The description of the trichotomous exposure score needs to be expanded with further justification added. The boundaries for the two non-zero exposure groups were selected so that the two groups would have equal numbers of cases and controls, and not on the basis of differences in exposure scores. No information is given as to the average or median exposure score for each of the different exposure time periods

and the maximum exposure score is not given for the different exposure time periods. The report emphasizes the dose-response relationship, but the information given is not sufficient to show that the two non-zero dose groups really have meaningfully different exposures. The data inconsistencies in exposure categories between Tables 6 and need to be addressed."

The peer reviewers describe the trichotomous exposure scores incorrectly. They state that "The boundaries for the two non-zero exposure groups were selected so that the two groups would have equal numbers of cases and controls..." This description implies that the exposure group cut points were selected to balance the number of persons in each category based on disease status. The cut point criteria, determined a priori, were designed to divide the exposed individuals in half, based on cumulative exposure scores for each time period of interest regardless of disease status. Disease status for each member of the exposed categories was not reviewed until category boundaries had already been assigned.

The exposure score cut point criteria, as discussed in the methods section, were developed to establish a standard method of assessing the exposed individuals based upon uncertainty regarding the existence of threshold exposure levels of Wells G and H water and adverse health effects.

An alternative method regarding the establishment of exposure group categories as suggested by the peer reviewers was attempted. A dose-response gradient was created combining water model exposure estimates with responses to water consumption interview questions to establish three new exposure groups. The lowest exposure group was comprised of persons who could not have received contaminated water from the well. The middle exposure group received contaminated water but primarily consumed store-bought beverages or bottled water. The high exposure group received contaminated water and used it as a primary beverage.

Results of analyses conducted for the exposure period during pregnancy supported our previous findings and suggested a positive association between having consumed contaminated tap water as a primary beverage and disease status. The magnitude of the association was similar to that of previous adjusted results for the same exposure time period. Only one case was identified as belonging to the middle exposure category as defined above resulting in unreliable statistical results for this exposure category.

Exposure value frequencies for cases and controls by time period are presented in table 10. Data inconsistencies in exposure categories between tables 6 and 10 have been corrected.

* Page 2, second full paragraph of the peer review comments states, " Some additional refinements in the water exposure assessment would be useful, but it would be more informative if examined as part of a dose-response gradient. The lowest exposure group might be no contaminated water, a middle group that received but probably did not drink contaminated water, and a third group that received and drank contaminated water."

This paragraph calls for re-analysis of the water distribution model as was described above.

* Page 2, third full paragraph of the peer review comments - This paragraph discusses the potential for exposure to Wells G and H for all study cases. It states, "It is not clear whether any of the cases diagnosed after 1979 had the potential for exposure to Wells G and H during pregnancy given when use of the wells ceased. Not enough information is presented to evaluate this. Some comment regarding the consequences of the [illegible] calendar time of potential exposure for cumulative exposure scores is warranted. It seems that since exposure is not possible after 1979, calculation of a cumulative exposure measure up to 1987 will dilute the exposure levels, for some subjects relative to those who could have been exposed throughout their pregnancies."

All cases had the potential for exposure at some point during their etiologic period, at the very least during the pre-

conception period. The calculation of cumulative exposure through 1987 although the wells were closed in 1979 does not serve to minimize exposures which occurred prior to the closure of the wells.

The computer program which calculated exposure assigned a value of zero to each month for which there was no possibility of contaminated water reaching the home of a subject. Exposure values were simply added once the values for each month of residence was assigned. Adding a series of zero values to a small number still results in that same small number. The number of zero exposure months per case therefore, has no diminishing effect upon exposure values.

* Page 2, fourth full paragraph - "The use of different exposure cut-points is potentially problematic in interpreting the results in Table 11. If exposure is expressed per unit time, the cut points and scales may be more comparable."

As a method of addressing the variability in exposure values which exist among exposure categories, peer reviewers suggested assessing exposure using normalized exposure scores representing exposure per unit time. When data were normalized, the effect was to narrow the range of potential exposure scores and thus minimize the effect of more extreme exposures while eliminating cumulative exposure effects. Normalized, unadjusted odds ratios

for exposure during pregnancy resulted in a positive association between disease status and exposure which was greater (O.R.=9.97, C.I.=1.22,81.22) than that of the non normalized (O.R.=5.70, C.I.=0.67, 48.25) results. The finding was statistically significant which was not the case in the non-normalized results. The normalized data, as explained in the text, confirmed the existence and direction of a possible association between water exposure and disease status but wider confidence intervals resulted in less stable statistical conclusions.

Table 11 presents odds ratios for exposure during different segments of the etiologic period. The different cut points represent cumulative exposure values which are based on differing time intervals of exposure. Tables 10 and 11 have been modified so that exposure is presented in reference to relative categories, not actual exposure values. The reader's attention is now more appropriately drawn to the statistical relationships associated with relative exposure rather than the numeric exposure values. The numeric values are more clearly explained within the context of the narrative.

* Page 2, fifth full paragraph - "A useful calculation might be to assess what proportion of cases are potentially attributable to consumption of contaminated water before and during pregnancy.

Does this exposure potentially account for a sizable fraction of the excess risk observed in Woburn?"

Attributable risks were not calculated because the odds ratios on which they are based, were extremely imprecise. The variability of the attributable risk estimates would be very great and, therefore, would not be particularly informative.

* Page 2, Control Selection - Peer reviewers were concerned whether the sampling frame for controls fully identified children who would have been eligible for the study had the control population been selected concurrently with case development. They state, "A key concern in this study, as in all case-control studies, is the suitability of the control group as a representative sampling from the study base that generated the cases. Some additional tabulations and discussion might help in judging the adequacy of controls. The overall strategy of sampling randomly from the entire community of Woburn is appropriate. School records provide a potentially useful sampling frame for generating the desired sample, but in spite of the detailed explanation of procedures, it is not clear whether the sampling frame for controls fully identifies children who would have been eligible for the study had it been done concurrently. That is, if the risk set had been fully enumerated at the time each case developed, how would that risk set compare to those who actually constituted the sampling frame for

controls. Potential concerns include selection of controls for preschool age children, children who attend private schools, and loss of potential controls due to having moved after the relevant diagnosis date. Beyond the question of whether the targeted controls are appropriate is the question of whether non-response may have distorted the patterns." They continue, "Because the water exposure model generates estimates based on both location of residence and time period of occupancy, it is important to be certain that the sample of controls is unbiased with respect to those factors. A more direct discussion of the potential for bias in control selection is warranted, even though it is not at all obvious that such bias has occurred. Along with random error, it is a key source of uncertainty in judging the validity of the study results."

As explained in the text, controls were selected from the graduating class year of Woburn Public School aged children to which the case belonged. By the date of control selection all study subjects were at least of elementary school age. This means that the complete public school cohorts of children diagnosed as pre-schoolers was available from the public school records at the time of control selection.

The school record cohort is somewhat different from the birth cohort. Use of a birth cohort would have created a much more difficult situation for tracking children than a cohort selected

from school records. School records are updated at least each school year and changes of address are recorded. If a person were to leave the Woburn Public School System, the address to which records are forwarded is often recorded allowing for the obtainment of a valid address outside of the Woburn area.
Not all cases were born in Woburn. Using a birth cohort as a source of controls would have required that a birth cohort for children born in the case's birth town would have to be obtained. This would have required searching for controls in other towns or other states based upon birth address alone, an extremely time consuming and impractical process.

Also lost from this cohort are those individuals who attended transferred to private schools. If a child of a Woburn resident never attended Woburn public Schools but began elementary school in a private facility, then they would not be represented in the cohort.

Control selection is therefore affected in that certain children who entered private schools early in their public school careers or moved from Woburn before entering school were not part of the control selection cohort. Children who may have entered private schools during high school would have had permanent record cards on file at the High School and would have been included in this project.

The control selection process is therefore not necessarily that which would have been used if controls were selected from cohorts concurrent with case diagnosis. It is difficult to assess what effect the loss of students attending private schools would have on the control population. Those that are lost from the control cohort population are more likely of higher socioeconomic status (SES). The public school cohort may therefore have been of slightly lower SES than the general Woburn population.

The water exposure model demonstrated the majority of water exposure to be in East Woburn although up to two thirds of Woburn received some contaminated water while the wells were functioning. East Woburn contains residences with a mix of SES. It is likely therefore that small shifts in the SES of the control cohort resulted in any difference in the likelihood of exposure to Wells G and H water when the public school cohort and the city-wide age specific cohorts are compared.

Confounding

* The peer reviewers state, "The two strategies for controlling confounding were to use a composite score or include all pre-selected covariates in a logistic regression model. An alternative not considered was selective inclusion of only the subset of individual variables that generated a change in the

coefficient for the variable of interest. In fact, there was little effort to identify the potential risk factors that actually appeared to be associated with childhood leukemia in these data (e.g., dental x-rays) as a restricted set of potential confounders to be controlled in the analysis. It appears that there was little confounding overall and it is quite possible that adjustment for one or two covariates would have been sufficient to control confounding without much loss of precision relative to the unadjusted results."

Peer reviewers suggested reconsidering the exposure-outcome relationship while controlling independently for each of the covariates showing in Table 22. Results of each analysis demonstrated adjusted odds ratios which were close to those obtained in the original composite covariate analyses (Table 8). For the period during pregnancy for example, the original adjusted composite covariate odds ratio value was 8.33 (C.I.= 0.73,94.67) while the odds ratio for the period during pregnancy adjusted for the effect of breast feeding revealed an odds ratio of 9.29 (C.I.=0.64,133.7). Exposure during pregnancy continued to demonstrate the strongest exposure relationship. This was most evident when results were adjusted for dental x-ray exposure during pregnancy (O.R.= 10.38, C.I.=0.72,149.2) compared to the composite adjusted odds ratio value (O.R.=8.33, C.I.=0.73,94.67).

To assure the most complete understanding of these covariates as potential confounders, a new composite covariate was created using the Tukey method as described in the text with the covariates listed in table 22 and also including the dental X-ray covariate. Adjusted odds ratios were similar to those presented on table 8. For the period before conception for example, the original composite covariate revealed an odds ratio of 2.61 (C.I.=0.47,14.37) while adjusting with a composite covariate based on the variables in table 22 revealed an odds ratio of 3.36 (C.I.=0.42,27.3) The exception was the odds ratio describing the relationship for exposure during pregnancy. The odds ratio for this relationship rose from the original adjusted value of 8.33 (C.I.=0.73, 94.67) to 148.4 (C.I.=1.29,17037.9).

Although the odds ratio is statistically significant, the instability of the statistical calculation, demonstrated by the large confidence intervals, warns that these results should be interpreted with caution. Adjusting for the main effects that demonstrated a statistical association has shown that the relationship between exposure during pregnancy and disease outcome continues to demonstrate a presence after adjustment for all reasonable covariates either plausible as indicated in the literature or possible as demonstrated in univariate analysis.

* Paragraph 1 - The peer reviewers comment that, "results of the study are comprehensively presented, including the needed

descriptive tabulations and unadjusted measures of association. Examination of risk for specific time windows is a very useful strategy. Although the imprecision of risk estimates is known to be a major source of uncertainty, the repeated notation of which measures of association are and are not statistically significant is unnecessary."

The text was trimmed to eliminate repetitive statements concerning significance.

* Paragraph 3 - "Some specific observations were not given as much attention or comment as they might deserve. There were relatively large (although non-statistically significant) differences in risk observed in Table 13 when accounting for types of leukemia. This table appears to suggest a very large effect for AML/CML cases on the risk (since the risk is elevated from 2.8 to 5.7 based solely on 4 more cases), which should be noted and interpreted in the text."

Peer reviewers note that AML/CML cases exert a strong effect on relative risk when included in the study population. Odds ratios for the ALL cases alone is 2.86 and goes up to 5.69 when AML/CML cases are included. Based on the fact that there are only four AML/CML cases, this small number of cases in these subtypes makes interpretation regarding the significance of this observation fairly limited.

* Paragraph 4 - "A more extensive effort to compile the literature specific to environmental exposures and childhood leukemia would be helpful in providing the context for these results. Although studies are cited in a general way, the evidence specifically on exposure to organic solvents during pregnancy and subsequent risk of leukemia in the offspring should be scrutinized, for example. Other peripherally relevant studies of these agents in relation to adult leukemias, birth defects and other adverse pregnancy outcomes, or health effects in general should be considered. A firm judgement of whether contaminated water caused the increased risk of leukemia is not possible but a better assessment of that possibility could be made based on more details of other relevant studies."

A review of the literature has revealed limited evidence specific to exposure to organic solvents or any other chemical agents and their effect during pregnancy and subsequent risk of leukemia. Little evidence in the literature means speculation regarding cause and effect is more open for interpretation.

SPECIFIC COMMENTS BY INDIVIDUAL REVIEWERS

The page numbers referred to in this portion of the document are the page numbers in the childhood leukemia report version for which the peer reviewers had comments.

Comments from David Savitz

Executive Summary

* Page 2 - The reviewer commented, "The general methods of control selection should be described."

Control selection has been discussed in more detail in the final version.

* Page 2 - The reviewer commented, "Selection of results for presentation solely on the basis of statistical significance rather than the interest or importance of the question is not a good strategy."

Results have been presented with more of a balance between significance and importance of the question.

* Page 3 - Peer reviewers commented, "Results for dose-response analysis should include estimates of relative risk, not just p-values for trend...The breast feeding results should be tempered given contradictory evidence in other studies."

Conclusions regarding the association between breast feeding and leukemia have been tempered from earlier drafts.

Introduction

* Page 4, paragraph 1 - The reviewer commented, "Is the absence of an unusual geographic pattern in childhood leukemia mortality discrepant with a geographic pattern to the exposure?"

Geographic patterns of leukemia incidence are not discrepant with the geographic patterns to exposure.

* Page 24 - The reviewer commented, "In the section on solvents, tobacco, etc., there needs to be a much clearer separation of evidence related to childhood leukemia versus adult leukemia. Current wisdom would suggest that the adult leukemia studies may have little relevance to risk factors for childhood leukemia."

Adult and childhood leukemia literature have been more clearly distinguished in this version of the document.

* Page 27 - The reviewer commented, "Some literature citations are needed in the first paragraph."

Appropriate citations have been added.

Methods

* Page 32 - The reviewer commented,"Matching in case-control studies does not control confounding but creates balanced groups that, when properly analyzed, make it easier to control confounding."

The text was adjusted to reflect this correction.

* Page 42 - "Did cases have a similar restriction to that described for controls with regard to when they had to have attended school?"

Cases and controls met the same school attendance criteria.

* Page 59 - The reviewer commented, "Trimesters are 13 rather than 15 weeks".

The misstatement regarding trimester length was corrected.

* Page 67 - The reviewer commented, "Should say 'lowest exposure group' not tertile."

The appropriate adjustment was made to the text as suggested.

* Page 69 - The reviewer commented, "In discussing confounding, it should be acknowledged that we actually know very little about determinants of childhood leukemia and that the list includes

many variables of potential interest which may well not be risk factors at all."

The discussion regarding confounding has been adjusted to explain that the variables of interest may in fact not prove to be risk factors.

* Page 70 - The reviewer commented, "How did you define 'significantly affect' in quantitative terms?"

The word "significantly" was removed from this portion of the narrative. The terms of the model were selected to provide the least complex model without affecting the odds ratio.

Results

* Page 72 - The reviewer commented, "What was the final effective control response proportion?"

There were nineteen eligible controls who did not participate in the study. Thirty seven eligible cases participated making a total of fifty six eligible controls. This means that 19/56 or 66% of the eligible controls responded.

* Page 73 - "Distinguish between ineligible (who should not be counted in calculation response proportion) and refusals (who should be counted)."

The text has been changed to distinguish between ineligibles and refusals in calculation of refusal rates.

* Page 74 - The reviewer commented, "In selecting parents, it is not clear whether distinctions were made for biological versus adoptive parents, and whether fathers and mothers had to be currently married for the father to be interviewed. This should be clarified in the Methods section."

This point is now explained more clearly in the text. All interviewed parents were biological parents. It was our intent to interview each parent regardless of marital status or length of residence with the study subject.

* Page 75 - The reviewer commented, "First full sentence: Something is wrong with this sentence."

The sentence has been corrected.

* Page 76, 8-10 lines from the bottom - Reviewers commented, "This is confusing and not quite correct in describing what an odds ratio is. It is the odds of exposure among cases versus

controls; nonexposed cases are the denominator of the odds of exposure among cases and similarly for the controls."

The description of an odds ratio has been corrected as suggested.

* Pages 83 and 86 - The reviewer commented, "Here and elsewhere that statistical tests are performed, it would be preferable to provide the actual p-value rather than to note only whether or not it was significant." They further comment, "Selection of statistically significant results for presentation is not a good strategy. Given the need to be selective since so many odds ratios were generated, those that are based on questions of interest and where prevalence is adequate to study would be the most important, regardless of whether an association was found. Also, there is no clear logic to the sequence of the presentation, making it hard to follow the flow of information. Environmental exposures might be grouped, for example."

Results of tests of significance have been provided in the narrative for associations which seem plausible in relation to disease outcome and in circumstances of statistical significance. In other instances, the reader is referred to tables for complete statistical results.

* Page 91, 92: The reviewer commented, "Presentation of a summary of results (not just in appendix) would be useful."

As there were very few positive responses to this series of questions, presenting a summary in the narrative portion of this report was not felt necessary.

* Page 92, ten lines from the bottom - The reviewer commented, "Which direction was the age association -- higher risk with older or younger maternal age?"

The children of older mothers were not at greater risk than those of younger mothers.

* Page 93 at the bottom - The reviewers commented, "Put employment information with occupation results."

The employment information was included with the occupational results but mentioned in this portion of the report as it was part of the univariate analyses.

* Page 94, paragraph 3 - The reviewer commented, "Why doesn't 6 exposed controls and 0 exposed cases generate an odds ratio of 0.00 ?"

The text was adjusted accordingly.

* Page 95, Bottom - The reviewer commented, "The low prevalence of breast feeding (presumably for any period of time) is so

notable that it calls into question the results. This might be mentioned in the discussion."

The discussion was adjusted accordingly.

* Page 99-102 - The reviewers commented, "The EMF results are made far more convoluted and confusing than they need to be. You could start by creating variables for specific times such as 'wire code at diagnosis' to analyze and avoid pools of homes without regard to time of occupancy. Given measurement data, there are three discreet groups: Very high current configuration, ordinary high current configuration, and everything else. You could group the lowest categories together and present the results for the other contrasts (OHCC vs. low, VHCC vs. low). It is not necessary (and is somewhat confusing) to present so many others."

With the amount of concern the community has expressed regarding EMF and its effect on leukemia incidence in Woburn, we are reluctant at this point to cut back on the level of detail presented in the text. We may choose to modify this section after the public comment period for this report.

Discussion

* Page 113 - "The contradictory literature on breast feeding and childhood cancer should be cited."

The literature regarding breast feeding and childhood cancer seems neither to support nor deny this relationship.

* Page 114, 4 lines from the bottom: - "Underground wiring is not assumed to have zero exposure but rather to be in the lowest group."

This comment was noted and the text was adjusted accordingly.

* Page 116 Bottom - "It is not clear why the observed distribution of wire code categories suggests that 'Woburn's neighborhoods are older...'."

The wire code categories themselves were not the indicators of community stability. Observations made and information learned while reviewing the demographics of this community lead to this conclusion. The text was adjusted to state this more clearly.

Responses to Dr. Stanek's specific comments:

* Page 19, lines 11 to 8 - The reviewer comments, "I don't see how the difference in the spike will result in the conclusion you attribute to it."

The limited medical attention this population receives would preclude the diagnosis of leukemia in early childhood. It may be that this population is not less susceptible to leukemia but that the medical attention required for diagnosis is simply not available. Leukemias would go undiagnosed and causes of death would be incorrect or unknown.

* Page 28, lines 3-5 and 35 line 4 - Reviewer Comments, "Awkward sentence structures and references for 1981 study need to be included."

References added and sentence structures were adjusted as suggested.

* Page 55, top paragraph - Reviewer comments, "The evaluation of the water model indicates that there will be a substantial miss-classification of exposure measures for individual subjects based on the test data. Such misclassification will bias risk estimates down. Does this deserve some discussion?"

The issue of water model misclassification has now been addressed in more detail throughout the text.

* Page 66, line 8 - The reviewer's comments, "I feel you should expand on your description of the model. You use a proportional hazards model to fit the conditional logistic regression model to

case/control status. The report never seems to state that clearly, nor to reference the connection between the analysis. This should be amplified. There is some discussion of this on page 71. However, you never write out a model equation, or reference the method (other than a general reference to the SAS program)."

A more thorough discussion of the model has been added to the text.

* Page 68, line 6 - The reviewer's comment, "Use of Tukey's suggestion for collapsing covariates may be reasonable. Additional information that would have been valuable is an evaluation of the sensitivity of the choice of coefficients for the collapsing, since the method seems somewhat at hoc.

More detail regarding the coefficient choices has been added to the section concerning confounding.

* Page 74, line 9 - The reviewer's comment, "Figure 1 is unclear. It would help to identify the interval for case detection that you
have used when plotting cases."

As stated in the text, each point on the X axis represents those cases diagnosed in that particular calendar year.

* Page 76, line 6 - "I don't find the ranges reported in table 7 of much value. Why not report medians, quartiles, or other descriptive statistics."

The ranges were reported to give the readers a sense of the potential exposure values that could be expected per unit time.

* Page 80, line 11 - The reviewer's comment, "I am missing the rationale for the trimester analysis, other than being another interesting thing to look at. Do you have any rationale for expecting a difference in one direction or another? It seems somewhat like a fishing expedition, and has principally descriptive value. What conclusions do you make from your analysis results?"

Examining exposure by trimester assesses the possibility that the fetus may have been more sensitive to environmental insult at different times throughout the pregnancy, particularly early in the pregnancy when fetal organogenesis proceeds most rapidly. If low levels of exposure were to have an effect on the fetus, it would likely happen at this most sensitive time.

* Page 83, lines 5-9 - The reviewer's comment - "Did you do any analysis to evaluate the sensitivity to your dose classification?"

The sensitivity of the dose classification has been addressed more extensively in this version of the document in the discussion of the Murphy Water Model.

* Page 85, line 6 to 4 - Reviewer's Comment, "None of the results are significant. The ALL results are weaker. You seem to be 'beating the drum' here."

The major focus of this investigation was the feasibility of water as the vehicle for human exposure. The survey instrument was designed with questions which serve as redundant verifiers of water consumption. We have included assessments of water exposure based on all of these variables to provide the maximum assurance that the best possible understanding of water exposure was used to assess cause and effect.

* Page 96, line 10 - Reviewer's Comment, "This is one of the few results statistically significant, but is under billed. More discussion is warranted. Do you consider it a 'chance' finding?"

As discussed in the text, this relationship, although statistically significant was based on six case mothers and two control mothers responding positively to the X-ray use question. The instability of the statistical calculation warns that these results be interpreted with caution and certainly their

likelihood as a 'chance' finding should not be ignored. Five of the eight mothers were not sure if a lead apron shield was used during the X-ray procedure. A more complete account of the use of lead shields during these procedures would provide insight regarding the amount of exposure likely to have occurred and therefore whether the X-ray exposure association suggest a plausible relationship to the onset of leukemia in offspring.

* Page 113, lines 15-19 - "More discussion (and possible analysis) of how breast feeding fits into the picture for incidence with the other variables is warranted."

This suggestion has been incorporated into the final report.

* Page 117, line 7 - Reviewer's comments, "Is table 13 the right table to reference?"

The table number has been corrected.

* Page 122, 2nd paragraph - Reviewer's comments, "I don't understand why the discussion brings in "sensitive individuals", or how this adds to the interpretation of the results."

The studies regarding TCE which are cited are occupationally based. Results show no association to cancer in this population. A worker population however, tends to be healthier than the

general population which includes more environmentally sensitive individuals such as children, elderly, and those too ill to work. It is difficult to determine if the effect or lack of effect from an exposure received by a healthy worker would be the same as that experienced by a person more sensitive to adverse outcome.

* Page 125, lines 12-18 - "I think this explanation is not very plausible. Are subjects equally aware of other risk factors? Wouldn't they have to be to bias the results negatively?"

Parents of cases who believe water consumption was the primary cause of the illness and are anxious to demonstrate this through our results could bias results toward a water association by overemphasizing their child's water consumption in the course of responding to interview questions. Parents do not need to be aware of other risk factors for this to occur. Their overemphasis of water exposure in the context of [illegible] responses to other questions could result in an artificially elevated association of the relationship between contaminated water consumption and disease outcome.

* Pages 126, line 8 - Reviewer's comments, "'...leukemia was greater for...' I think this should be toned down."

This suggestion has been incorporated into the document by the addition of qualifying language which more clearly explains the

limitations of the conclusions. In general, the conclusions are based upon imprecise estimates of leukemia risk because of the small number of subjects. This imprecision indicates that the magnitude of an effect is indefinite. When statistical significance is not achieved, an estimate of risk that appears to be increased may in fact be due to the statistical variability inherent with estimates based on small numbers. However, considering these limitations, different approaches were taken in the analysis to assess the validity of an association between exposure to water from wells G and H and risk of leukemia. The results consistently were in the direction of an association between exposure and risk, although the magnitude of the association remains unclear.

Responses to Nancy Kim's Comments

* Page 49, Paragraph 1 - Reviewer's comment: "'It was assumed that leakage flow was uniformly distributed through the system.' Given this assumption, what is its basis and could any errors be introduced?"

This assumption was made based upon the fact that there is no good historic data which would indicate where major leaks in the Woburn water system are located and that the water model validation study showed that despite the fact that leakage data was not included in the model, it predicted water distribution

within an acceptable range. Dr. Murphy's March 1986 report states, 'In 1980 the Pipe Line Testing Service Company performed a water leakage study to determine the extent of any leakage in the water distribution system. Their report indicated very substantial leakage totalling 1.7 million gallons per day. Although there is some doubt concerning the accuracy of this amount, it was clear that Woburn had a serious leakage problem. Since residential users are billed on a flat rate rather than from meter readings, there is only a poor description of the areal distribution of water use in Woburn at the present time.'"

In Dr. Murphy's 1990 report describing his revised water distribution model he states, 'Although there was some evidence of significant leakage flows,...(he refers to the above statement)...because of their uncertain location no attempt was made to include the spatial distribution of leakage in modeling the user demand distribution.'"

If leakage flow was not uniformly distributed, certain neighborhoods would loose water to the outside of the system faster than others and the predicted values for contaminated water distribution would be incorrect. It is not possible to predict in which direction the error would skew the results unless the specific points of major leaks which were occurring at the time of the wells' operation could be identified. If leaks in the system occurred along the path between the contaminated

wells and the residences resulting in loss of contaminated water from the system, then the Murphy model may be overestimating, the level of contaminant concentration at the tap. If leaks occurred predominantly along the path of the uncontaminated water lines, then more contaminated water could have flowed into the system to meet household demands. There was no evidence to suggest that leaks predominated in any one particular area of the water supply.

* Page 49, Paragraph 1 - Reviewer's comment: "'...water consumption was estimated by applying the value of 370 gallons per day to each residential...' What is the basis for this assumption?"

The hydrogeologist who developed the water model used in Woburn consulted a Woburn water supply requirements [illegible] 1970 which indicated that the residential and com[illegible]mption in Woburn was 85 gallons per capita per day [illegible]. It also indicated that the user demand was gradually increasing with time to 100 gallons per day by 1985. The census data of 1960 showed an average of 3.7 people per household and so a "good" estimate of 370 gallons per day per residential water meter is based on this data.

* Page 52, Paragraph 1 - Reviewer's comment: "'Root mean square head difference was 6.2 feet.' Can you give the reader a

qualitative idea as to the importance of the error level and root mean square head difference?"

Dr. Murphy, the hydrogeologist who developed the water model stated that although there is a mathematical relationship between root mean square head difference and flow rate in a water model, the data necessary to fully understand this relationship for Woburn's water supply was not available.

The purpose of the Woburn Water Model however, was to predict the concentrations of contaminated water distributed among residential areas served by Woburn public water. Data describing water consumption patterns and flow rates over time were sufficient to develop a good understanding of the distribution of water from Wells G and H.

The best way to predict the error level of the model then, was through a validation analysis which measured the predictability of the contaminated water concentration along the system. The validation analysis, summarized in this report, shows that the model was able to predict contaminated water concentrations within a range considered sufficient by water model peer reviewers.

* Page 54, paragraph 1 - Reviewer's comment: "'Overall, 72 percent of the field test predictions were within 0.2 of the observed values.' Should the value 0.2 have units?"

The units here are 0.2 ppm.

* Page 89, Paragraph 1 - Reviewer's comment: "'Once again, odds ratios demonstrate no association between occupational categories most frequently reported and childhood leukemia incidence.' I don't disagree with this statement although I was troubled in reading this section by what appears to be inconsistencies as to which comparisons are pointed out and discussed and which were ignored. The selection is not statistical significance, but what is it? For example, is it an odds ratio of 1.5 and that is why the fathers in the durable goods manufacturing industry were discussed and why mothers in the clerical category weren't, e.g. 1.98 (0.63, 4.72) and 1.40 (0.50, 3.97)?"

Main effects with ten or more positive responses and with an odds ratio of 1.5 or greater were included in the model and discussed in the text. Main effects which are commonly associated with cancer incidence or of particular interest to the Woburn community were also highlighted.

* Page 99, Paragraph 3 - Reviewer's comment: "'Forty-six of the seventy-three study subjects addresses (63%) were classified as

OLCC.' I may have missed it, but I couldn't find the definition of OLCC, VHCC, etc.

OLCC is an acronym for "ordinary low current configuration", VHCC is an acronym for "very high current configuration". The full wording has been inserted into the text here and was discussed in detail in the methods section.

* Page 108, Sentence 3 - Reviewer's comment: "'Although - confidence intervals are extremely wide for odds ratios of individual pregnancy strata, a test for trend is significant suggesting the existence of a dose-response relationship between wells G and H water and childhood leukemia for exposure during pregnancy.' I am uncomfortable with this and some other statements which focus on the importance of the dose-response relationship, given the description of the exposure data. However, if additional data clearly show a difference in exposure between the two non-zero dose groups, my concern will be diminished."

As a result of the peer review of this document, the text has been edited to include more qualifying language regarding the associations between water exposure and leukemia incidence. The dose-response relationship referred to in this particular comment has been qualified as part of this process. Qualifying language added to the conclusions states, "In general, the conclusions are

based upon imprecise estimates of leukemia risk because of the small number of subjects. This imprecision indicates that the magnitude of an effect is indefinite. When statistical significance is not achieved, an estimate of risk that appears to be increased may in fact be due to the statistical variability inherent with estimates based on small numbers. However, considering these limitations, different approaches ere taken in the analysis to assess the validity of an association between exposure to water from Wells G and H and risk of leukemia. The results consistently were in the direction of an association between exposure and risk, although the magnitude of the association remains unclear."

* Page 109, Sentence 1 - Reviewer's comment: "'The tap water question must be interpreted with caution however as respondents were likely to be aware of the community's concerns regarding ill health and exposure to contaminated public water.' This is a reasonable qualifier, but these types of qualifications are not carried over into the executive summary."

The executive summary has now been qualified to the same degree as the main report.

* Page 116, Paragraph 2 - Reviewer's comment: "'Odds ratios resulting from the relationship between wire code configuration and childhood leukemia do not support a positive association

between wire code and leukemia incidence.' As mentioned previously, the criteria for stating when odds ratios, not statistically significant, are considered a positive association and when they are not need to be defined."

The criteria for the presentation of odds ratios in the text are explained in the response to comments for page 89, paragraph 1, above.

* Page 123, Paragraph 1 - Reviewer's comments, "'Alternatively, fetal susceptibility to potential drinking water contaminants could reflect high dose exposure. Regular maternal ingestion could lead to sustained blood levels or even concentrations in fetal tissues.' This sentence should be rewritten so the meaning is clear. In fact, the statement may not be necessary, given the sentence that follows."

The sentence "Alternatively, fetal susceptibility to potential drinking water contaminants could reflect high dose exposure." has bee removed from the text.

* Page 126, Paragraph 1 - Reviewer's comments, "'...showed a significantly positive relationship to the amount of water mothers consumed.' Would the following statement be more accurate? 'Showed a significantly positive relationship to the contaminant levels in the water delivered to the households.'"

This sentence has been adjusted as suggested.

* Page 128, Paragraph 1 - Reviewer's comments, "'ongoing cancer incidence surveillance will provide information to suggest whether control of environmental hazards in Woburn has coincided with a more predictable rate of childhood leukemia incidence.' Isn't it more accurate to refer to the control of specific contaminants in drinking water rather than environmental hazards?"

The reviewer is correct in stating that the main issue is the control of drinking water contaminants.

* Table 10 - "Some of the data in this table are not consistent with the data in Table 6. The number of cases in the never exposure category for the exposure time period from 2 years before conception diagnosis is 4 in table 6; however, the number of cases with the 0.00 exposure score in table 10 for the same exposure period is 3. A similar problem occurs with the exposure time period "during pregnancy". In addition, the third exposure category for most of the time periods has a '<' sign before it; the sign should be '>'. In table 11, some of the signs are omitted and need to be added."

The study subject frequencies in tables 6 and 10 have been corrected. In table 6 the exposure time period from 2 years

before conception to case diagnosis has 3 cases in the "Never" category and 16 cases in the "Ever" category. In table 10 for the same exposure time period there are 3 cases in the "Never" exposure category, 9 cases in the "Least" exposure category, and 7 cases in the "Most" exposure category.

For the exposure time period during pregnancy in table 6 there are 9 cases in the "Never" exposure category and 10 cases in the "Ever" exposure category. In table 10 for the same time period there are 9 cases in the "Never" exposure category, 3 cases in the "Least" exposure category, and 7 cases in the "Most" exposure category.

Tables 10 and 11 have been modified so that exposure categories are defined as "Never", "Least", and "Most" to alleviate confusion regarding the interpretation of differences in exposure values.

APPENDIX III

Histology Codes

APPENDIX III

ICD-O (International Classification of Diseases for Oncology) codes used to define leukemia as part of the case definition for this study.

Histology codes/Primary site 169.1

ICD-O 0 9800-9940, 9951

APPENDIX IV

Case Contact Letter for Families who Participated in the MDPH 1981 Childhood Leukemia Study

The Commonwealth of Massachusetts
Executive Office of Health and Human Services
Department of Public Health
150 Tremont Street
Boston 02111

William F. Weld
Governor
Charles D. Baker
Secretary
David H. Mulligan
Commissioner

Date

Mr. and Mrs. (case family name)
27 N. Maple Street
Woburn, MA 01801

Dear Mr. and Mrs. :

I am writing to you today to enlist your assistance in the Massachusetts Department of Public Health's (MDPH) ongoing investigation of the elevated rate of childhood leukemia in Woburn. We greatly appreciated your past cooperation and hope that you will participate in the continued investigation of this important public health concern.

While most families of childhood leukemia cases have been interviewed by MDPH between 1981 and 1987, our present goal is to interview all case families with a more comprehensive interview than was previously used. We will be examining medical, biologic, genetic and environmental factors that were not identified at the time of the 1981 study and so were not asked during the earlier interview.

Your participation will involve an interview to be held in your home or at a convenient place of your choosing. The interview will take approximately one hour and all responses as well as names of all participants will be kept strictly confidential. Questions will be about your work and residential histories and you and your child's hobbies and medical histories. In order for us to complete the interview fully we ask that both parents be present, if possible.

We want you to understand your cooperation is completely voluntary and you have the right to withdraw from the interview at any time. No data on individuals will be revealed or reported that would identify you to anyone outside this project.

An interviewer from the Department of Public Health will contact you within ten days to invite your participation and arrange a convenient time to conduct the interview.

The success of the study depends upon obtaining information from everyone. Your cooperation is important and greatly appreciated. We feel the information you provide us may greatly contribute to a better understanding of childhood disease in Woburn. If you have any questions or concerns about this study, please feel free to contact me at (617) 727-7170.

Sincerely,

Robert S. Knorr, Ph.D.
Chief, Environmental
Epidemiology Unit
The Division of Environmental
Epidemiology and Toxicology

APPENDIX V

Case Contact Letter for Families who were not Part of the MDPH 1981 Childhood Leukemia Study

The Commonwealth of Massachusetts
Executive Office of Health and Human Services
Department of Public Health
150 Tremont Street
Boston 02111

William F. Weld
Governor
Charles D. Baker
Secretary
David H. Mulligan
Commissioner

Date:

Mr. and Mrs. (case family name)
Street Address
Woburn, MA 01801

Dear Mr. and Mrs. :

I am writing to you today to enlist your assistance in the Massachusetts Department of Public Health's (MDPH) ongoing investigation of the elevated rate of childhood leukemia in Woburn. We hope that you will participate in the continued investigation of this important public health concern.

While most families of childhood leukemia cases have been interviewed by MDPH between 1981 and 1987, our present goal is to interview all case families with a more comprehensive interview than was previously used. We will be examining medical, biologic, genetic and environmental factors that were not identified at the time of the 1981 study and so were not asked during the earlier interview.

Your participation will involve an interview to be held in your home or at a convenient place of your choosing. The interview will take approximately one hour and all responses as well as names of all participants will be kept strictly confidential. Questions will be about your work and residential histories and you and your child's hobbies and medical histories. In order for us to complete the interview fully we ask that both parents be present, if possible.

We want you to understand your cooperation is completely voluntary and you have the right to withdraw from the interview at any time. No data on individuals will be revealed or reported that would identify you to anyone outside this project.

An interviewer from the Department of Public Health will contact you within ten days to invite your participation and arrange a convenient time to conduct the interview.

The success of the study depends upon obtaining information from everyone. Your cooperation is important and greatly appreciated. We feel the information you provide us may greatly contribute to a better understanding of childhood disease in Woburn. If you have any questions or concerns about this study, please feel free to contact me at (617) 727-7170.

Sincerely,

Robert S. Knorr, Ph.D.
Chief, Environmental
Epidemiology Unit
The Division of Environmental
Epidemiology and Toxicology

APPENDIX VI

Control Family Contact Letter

The Commonwealth of Massachusetts
Executive Office of Health and Human Services
Department of Public Health
150 Tremont Street
Boston 02111

William F. Weld
Governor
Charles D. Baker
Secretary
David H. Mulligan
Commissioner

Date

Mr. and Mrs. Control
Street Address
Woburn, MA 01801

Dear Mr. and Mrs. Control:

I am writing to you today to enlist your assistance in the Massachusetts Department of Public Health's (MDPH) ongoing investigation of the elevated rate of childhood leukemia in Woburn. Your cooperation is important and will be greatly appreciated for the continued investigation of this important public health concern.

This study is being conducted among a sample of persons chosen at random who attended Woburn schools. Your daughter/son has been one of those selected to be included in this study.

Your participation will involve an interview to be held in your home or at a convenient place of your choosing. The interview will take approximately one hour and all responses as well as names of all participants will be kept strictly confidential. Questions will be about your work and residential histories and you and your child's hobbies and medical histories. In order for us to complete the interview fully we ask that both parents be present, if possible.

We want you to understand your cooperation is completely voluntary and you have the right to withdraw from the interview at any time. No data on individuals will be revealed or reported that would identify you to anyone outside this project.

An interviewer from the Department of Public Health will contact you within ten days to invite your participation and arrange a convenient time to conduct the interview.

The success of the study depends upon obtaining information from everyone. Your cooperation is important and greatly appreciated. We feel the information you provide us may greatly contribute to a better understanding of childhood disease in Woburn. If you have any questions or concerns about this study, please feel free to contact me at (617) 727-7170.

Sincerely,

Robert S. Knorr, Ph.D.
Chief, Environmental
Epidemiology Unit
The Division of Environmental
Epidemiology and Toxicology

APPENDIX VII

Contact Letter for Families with an Unlisted Telephone Number

The Commonwealth of Massachusetts
Executive Office of Health and Human Services
Department of Public Health
150 Tremont Street
Boston 02111

William F. Weld
Governor
Charles D. Baker
Secretary
David H. Mulligan
Commissioner

Date:

Subject Family
Street Address
Woburn, MA 01801

Dear Mr. and Mrs. :

On (Date) I sent you a copy of the enclosed letter asking for your assistance in the ongoing investigation of the elevated rate of childhood leukemia in Woburn. Since then my office has learned that your telephone number is unlisted.

We are still very much interested in contacting you to invite your participation in our study. Could you please call Mr. Kevin Costas at (617) 727-7170 to arrange a convenient time to be interviewed.

Sincerely,

Robert S. Knorr, Ph.D.
Chief, Environmental
Epidemiology Unit
The Division of Environmental
Epidemiology and Toxicology

APPENDIX VIII

Consent Form Granting MDPH Permission

to Interview Study Subject Family

Michael S. Dukakis
Governor

Philip W. Johnston
Secretary

borah Prothrow-Stith, M.D.
Commissioner

The Commonwealth of Massachusetts
Executive Office of Human Services
Department of Public Health
150 Tremont Street
Boston 02111
617-727-2700

WOBURN CHILDHOOD HEALTH STUDY

CONSENT TO BE INTERVIEWED

I have been asked by a researcher from the Massachusetts Department of Public Health (MDPH) to participate in a health interview. I understand that I would be asked to answer health questions to assist in the understanding of childhood disease in Woburn. I have agreed to answer questions about my work and residential histories, and my child _____'s and my hobbies and medical histories. I understand that my cooperation is completely voluntary and that I have the right to withdraw from the interview at any time.

All medical information and other personal data obtained in this interview shall be kept confidential in accordance with the laws and regulations of the Commonwealth of Massachusetts relating to confidentiality and privacy. I understand also that no use will be made of information that would identify me, my child, or any of our relatives to anyone other than those researchers working for this study. If I have any questions regarding this study or my participation in it either now or at any time in the future, I am free to contact Dr. Robert Knorr at (617) 727-7170.

_____ _____
Signature Date

_____ _____
Signature Date

APPENDIX IX

Consent Form Granting MDPH Permission

to Review Study Subject Medical Records

The Commonwealth of Massachusetts
Executive Office of Human Services
Department of Public Health
150 Tremont Street
Boston 02111
617-727-2700

Michael S. Dukakis
Governor

Philip W. Johnston
Secretary

borah Prothrow-Stith, M.D.
Commissioner

WOBURN CHILDHOOD HEALTH STUDY

AUTHORIZATION FOR DISCLOSURE OF MEDICAL INFORMATION

Patient's Name:_____ Date of Birth:_____

I have received a letter explaining the purpose of this study. I understand this study aims to explore possible environmental causes of illness and that my participation is completely voluntary. I also understand that I may revoke this authorization at any time.

I agree to cooperate in this study by allowing my medical records, including identifying information, to be inspected and/or photo-copied by authorized research personnel from the Massachusetts Department of Public Health in the course of carrying out this important project. This authorization covers all medical treatment and history of illness or related information.

It is my understanding that all information collected will be kept strictly confidential in accordance with the laws and regulations of th Commonwealth of Massachusetts relating to confidentiality and privacy, and will be used for research purposes only. I understand also that no use will be made of information that would identify me or any of my relatives to anyone other than those researchers working for this study.

If at any time I wish to discuss my participation in this study with one of the researchers, I may contact Dr. Robert Knorr at (617) 727-7170.

I hereby authorize the _____ Hospital/Clinic to release information from my medical records to the Massachusetts Department of Public Health.

_____ _____

signature date

APPENDIX X

The Study Questionnaire

INDEX CHILD'S STUDY NUMBER _____

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
THE DIVISION OF ENVIRONMENTAL
EPIDEMIOLOGY AND TOXICOLOGY

WOBURN CHILDHOOD HEALTH STUDY QUESTIONNAIRE

MOTHER'S QUESTIONNAIRE

INDEX CHILD'S STUDY NUMBER: _____

Sex of (Index Child) 1-Male 2-Female

PART I: INTRODUCTION
SECTION A: IDENTIFYING INFORMATION

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
THE DIVISION OF ENVIRONMENTAL
EPIDEMIOLOGY AND TOXICOLOGY

WOBURN CHILDHOOD HEALTH STUDY QUESTIONNAIRE

1) INDEX CHILD'S NAME AND ADDRESS: _____

_____

_____

2) INTERVIEWEE'S NAME AND ADDRESS: _____

_____

_____

3) INTERVIEWEE'S TELEPHONE #: _____

4) RELATIONSHIP OF INTERVIEWEE TO INDEX CHILD: _____

5) DATE OF INTERVIEW: _____

"Hello, my name is _____. I am representing the Massachusetts Department of Public Health and am here with regard to our appointment for the completion of the Woburn Childhood Health Study Questionnaire. May I come in?"

"As you may be aware, this study is being conducted among a sample of children chosen at random from the general population. Your child has been one of those selected to be included in this study. Questions will be mainly about your work history, where you have lived, and you and your child's medical histories and hobbies. Did you have a chance to review the letter I sent you describing this study?"

I.D. #_____

IF YES: "Do you have any specific questions?"

"Let me remind you that your cooperation is completely voluntary. You have the right to withdraw from the interview at any time. All information collected in this study will be locked and kept in the strictest confidence in accordance with Massachusetts General Laws. No use will be made of information that would identify you to anyone outside this project."

IF NO: "Let me read it to you." [READ LETTER]

"Before we begin, could you please sign this consent form which indicates that you understand the purpose of this study and your rights as a participant as I have just reviewed them with you."

"Feel free to take as much time as you need to answer the questions. Some of them concern events which took place many years ago and we would like you to answer them as completely and carefully as you can. If there are any questions you do not fully understand, please let me know. If you are unsure of an answer to a specific question, please say so."

I.D. #_____

SECTION B: DEMOGRAPHIC INFROMATION

I'd like to start by asking some questions about (Index Child's) background.

1) What is (Index Child's) date of birth?

(Month, day, year)_____

2) Where was (Index Child) born?

City_____

State or Country_____

3) Which of the following terms best describes (Index Child's) racial background?

1-Hispanic
2-White
3-Black
4-Asian
5-American Indian
6-Other (specify_____)
8-Unknown
9-Refused to answer

4) Which of the following terms best describes (Index Child's) religious background?

1-Roman Catholic
2-Protestant (specify_____)
3-Jewish
4-Other (specify_____)
8-Unknown
9-Refused to answer

PART II: RESIDENTIAL HISTORY

Now, I would like to ask you about residences you and (Index Child) have lived in. We will begin with the residence you lived in during _____ [MONTH AND YEAR OF DIAGNOSIS OF CASE] and work back to the residence you lived in during _____ [MONTH AND YEAR-2 YEARS PRIOR TO CONCEPTION OF INDEX CHILD].

[IF INDEX CHILD IS ADOPTED OR LIVED WITH A GUARDIAN, THEN ASK FOR THE RESIDENCES THE CHILD MAY HAVE LIVED IN PRIOR TO LIVING WITH INTERVIEWEE]

IDENTIFICATION NUMB_____

RESIDENTIAL HISTORY

RESID. NO.	a. WHAT WAS THE ADDRESS OF THE _____ (1ST, 2ND, ETC.) RESIDENCE?	b. FROM WHAT YEAR TO WHAT YEAR WAS THAT?	c. HOW WAS THE WATER SUPPLIED TO THIS RESIDENCE?	d. WHILE LIVING IN THIS HOUSEHOLD DID (INDEX CHILD) DRINK MOSTLY...
1	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
2	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
3	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
4	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
5	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown

IDENTIFICATION NUMBER _____

RESIDENTIAL HISTORY CONTINUED

f.

DID THIS RESIDENCE HAVE ANY OF THE FOLLOWING FEATURES?
PLEASE RESPOND AFTER EACH ITEM.

RESID. NO.	c. HOW MANY PEOPLE SMOKED CIGARETTES, CIGARS OR PIPES IN THAT HOUSEHOLD DAILY?	
1	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused 9 Unknown How many times in total?_____ ___use of "no-pest" strips ___attached garage
2	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused 9 Unknown How many times in total?_____ ___use of "no-pest" strips ___attached garage
3	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused 9 Unknown How many times in total?_____ ___use of "no-pest" strips ___attached garage
4	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused 9 Unknown How many times in total?_____ ___use of "no-pest" strips ___attached garage
5	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused 9 Unknown How many times in total?_____ ___use of "no-pest" strips ___attached garag

7.09

IDENTIFICATION NUMBER_____

RESIDENTIAL HISTORY

RESID. NO.	a. WHAT WAS THE ADDRESS OF THE _____ (1ST, 2ND, ETC.) RESIDENCE?	b. FROM WHAT YEAR TO WHAT YEAR WAS THAT?	c. HOW WAS THE WATER SUPPLIED TO THIS RESIDENCE?	d. WHILE LIVING IN THIS HOUSEHOLD DID (INDEX CHILD) DRINK MOSTLY...
6	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
7	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
8	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
9	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
10	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown

IDENTIFICATION NUMBER _____

RESIDENTIAL HISTORY CONTINUED

RESID. NO.	c. HOW MANY PEOPLE SMOKED CIGARETTES, CIGARS OR PIPES IN THAT HOUSEHOLD DAILY?	f. DID THIS RESIDENCE HAVE ANY OF THE FOLLOWING FEATURES? PLEASE RESPOND AFTER EACH ITEM.
6	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying ___use of "no-pest" strips How many times in total?_____ ___attached garage ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused How many times in total?_____ 9 Unknown
7	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying ___use of "no-pest" strips How many times in total?_____ ___attached garage ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused How many times in total?_____ 9 Unknown
8	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying ___use of "no-pest" strips How many times in total?_____ ___attached garage ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused How many times in total?_____ 9 Unknown
9	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying ___use of "no-pest" strips How many times in total?_____ ___attached garage ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused How many times in total?_____ 9 Unknown
10	# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying ___use of "no-pest" strips How many times in total?_____ ___attached garage ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds 8 Refused How many times in total?_____ 9 Unknown

PART III: OCCUPATIONAL INFORMATION

SECTION A: OCCUPATIONAL HISTORY

Now, I have some questions about your occupational history starting with the job you held during _____ [MONTH AND YEAR OF DIAGNOSIS OF CASE] back to the job you held during _____ [MONTH AND YEAR TWO YEARS PRIOR TO CONCEPTION OF INDEX CHILD].

We are looking for employer, job title, nature of the work, duration and potential exposures. Please be sure to note if there were any job title or type of work changes within the same company. [NOTE: PROMPT THIS AT THE END OF EACH JOB DESCRIPTION]

IDENTIFICATION NUMBER _____

OCCUPATIONAL HISTORY

JOB #	a. WHAT IS/WAS YOUR (____'S) EMPLOYER'S NAME & ADDRESS?	b. WHAT KIND OF BUSINESS WAS IT?	c. WHAT WAS THE TITLE OR NAME OF THAT JOB?
1	UNKNOWN...9	UNKNOWN...9	UNKNOWN...
2	UNKNOWN...9	UNKNOWN...9	UNKNOWN...
3	UNKNOWN...9	UNKNOWN...9	UNKNOWN...
4	UNKNOWN...9	UNKNOWN...9	UNKNOWN...
5		UNKNOWN...9	UNKNOWN...

IDENTIFICATION NUMBER: _____

OCCUPATIONAL HISTORY CONTINUED

JOB #	c. WHAT WAS THE NATURE OR TYPE OF WORK YOU DID?	f. WHAT MONTH & YEAR DID THIS JOB START?	g. WHAT MONTH & YEAR DID THIS JOB END?	h. DID YOU DRINK BOTTLED WATER AT THIS JOB?	i. DURING THAT JOB DID YOU OR WERE YOU EXPOSED (ask individually)
1		___/___ MO YR	___/___ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
2		___/___ MO YR	___/___ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
3		___/___ MO YR	___/___ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 FUMES 1 2 RADIATION 1 2 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
4		___/___ MO YR	___/___ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO CHEMICALS 1 2 FUMES 1 2 RADIATION 1 2 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
5		___/___ MO YR	___/___ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO CHEMICALS 1 2 FUMES 1 2 RADIATION 1 2 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		

JOB #	a. WHAT IS/WAS YOUR (____'S) EMPLOYER'S NAME & ADDRESS?	b. WHAT KIND OF BUSINESS WAS IT?	c. WHAT WAS THE TITLE OR NAME OF THAT JOB?
6	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9
7	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9
8	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9

JOB #	g. WHAT WAS THE NATURE OR TYPE OF WORK YOU DID?	h. WHAT MONTH & YEAR DID THIS JOB START?	i. WHAT MONTH & YEAR DID THIS JOB END?	j. DID YOU DRINK BOTTLED WATER AT THIS JOB?	k. DURING THAT JOB DID YOU USE OR WERE YOU EXPOSED TO: (ask individually)
6		___/___ MO YR UNKNOWN...9 UNK...9	___/___ MO YR UNK...9	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
7		___/___ MO YR UNKNOWN...9 UNK...9	___/___ MO YR UNK...9	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
8		___/___ MO YR UNKNOWN...9 UNK...9	___/___ MO YR UNK...9	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:

I.D. #_____

SECTION B: EXPOSURE HISTORY

I am now going to read you a list of chemicals. [SHOW LIST TO PARENT] Please tell me if you can remember ever using or being exposed to any of these chemicals in any of your jobs. If you are unsure, please say so. Were you exposed to (READ LIST):

CHEMICAL NAME	a. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	b. DURING WHICH DATES WERE YOU EXPOSED? FROM M/__ Y__ TO M/__ Y__	c. AT WHICH JOBS WERE YOU EXPOSED TO THIS CHEMICAL?	HOW WERE EX... 1-ALWA... 2-SO... 3-RA...
1) COKE OVEN EMISSIONS	____	M/__ Y__ M/__ Y__	_____	____ 1-AL... 2-SOME 3-RAR...
2) LEAD	____	M/__ Y__ M/__ Y__	_____	____ 1-ALW... 2-SO... 3-RA...
3) POLYVINYL CHLORIDE	____	M/__ Y__ M/__ Y__	_____	____ 1-A... 2-SOM... 3-RA...
4) CHLOROFORM	____	M/__ Y__ M/__ Y__	_____	____ 1-ALW... 2-S... 3-R...
5) IONIZING RADIATION	____	M/__ Y__ M/__ Y__	_____	____ 1-A... 2-SO... 3-R...
6) PESTICIDES	____	M/__ Y__ M/__ Y__	_____	____ 1-AL... 2-... 3-...
7) HERBICIDES	____	M/__ Y__ M/__ Y__	_____	____ 1-... 2-S... 3-...
8) SOLVENTS	____	M/__ Y__ M/__ Y__	_____	____

I.D. #

Did you ever use or were you ever exposed to any of these specific chemicals, while working?

CHEMICAL NAME	a. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	b. DURING WHICH DATES WERE YOU EXPOSED? FROM M/ Y	TO M/ Y	c. AT WHICH JOB WERE YOU EXPOSED TO THIS CHEMICAL?	d. HOW OFT WERE YO EXPOSE 1-ALWAYS 2-SOMET 3-RAREL
9) CARBONTETRACHLORIDE	____	M/ Y	M/ Y	_____	1-ALWAY 2-SOMET 3-RAREL
10) BENZENE	____	M/ Y	M/ Y	_____	1-ALWAY 2-SOMET 3-RAREL
11) TOLUENE	____	M/ Y	M/ Y	_____	1-ALWA 2-SOME 3-RARE
12) XYLENE	____	M/ Y	M/ Y	_____	1-ALWA 2-SOME 3-RARE
13) TRICHLOROETHYLENE	____	M/ Y	M/ Y	_____	1-ALW 2-SOME 3-RARI
14) METHYLENE CHLORIDE	____	M/ Y	M/ Y	_____	_____

PART IV: MEDICAL HISTORY

SECTION A: CHILD'S MEDICAL HISTORY

The next section of the questionnaire will focus on questions regarding (Index C
medical history.

I.D. #_____

O.K., now I'm going to read through a list of specific illnesses which I'd like to (Index Child) ever had.

ILLNESSES Has (Index Child) ever had:	a. What were the dates of its occurrence? (Months/Year) FROM	TO	b. What type of treatment did he/she have?	c. What we treatmen (Mon FROM
1) Infectious mononucleosis? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
2) Allergies? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
Has he/she ever had:				
3) Asthma? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
4) Chicken pox? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
5) Rubella (German measles)? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
Has he/she ever had:				
6) Rubeola (Measles)? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y
7) Mumps? 1-Yes 8-Refused to answer 2-No 9-Unknown	M/ Y	M/ Y		M/ Y

I.D. #_____

ILLNESSES (continued)	What were the dates of its occurrence? (Months/Year) FROM	TO	What type of treatment did he/she have?	What were the treatment dates? (Months/Year) FROM	TO
Has he/she ever had:					
8) A Shigella infection? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M/__
9) A Salmonella infection? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M/__
10) Aplastic anemia, which is a bone marrow defect? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M/_
Has (Index Child) ever had:					
11) Cytomegalovirus, which is also called salivary gland virus disease or inclusion disease? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M/
Has he/she ever had:					
12) A urinary tract infection? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M
13) Cancer? 1-Yes 8-Refused to answer 2-No 9-Unknown	___M/___Y	___M/___Y	_____	___M/___Y	___M

IF NO, SKIP TO PAGE 21, QUESTION 14

I.D. #_____

IF YES:

13a) What kind of cancer has he/she had?
[DO NOT READ LIST, CIRCLE APPROPRIATE NUMBER]

01. Lung	09. Hodgkins/Lymphoma
02. Breast	10. Kidney
03. Colon/Rectal	11. Liver
04. Pancreas	12. Cervix
05. Prostate	13. Other Female Organs
06. Stomach	14. Other Site (specify_____)
07. Bladder	15. Unknown
08. Leukemia	16. Refused to answer

b. What was the date of diagnosis for the (TYPE # ____) cancer?: ____M/____D/____

c. What was the date of first onset of symptoms for this cancer? ____M/____D/____

[IF APPROPRIATE, REPEAT QUESTIONS FOR A 2ND CANCER TYPE]

d. What was the date of diagnosis for the (TYPE # ____) cancer?: ____M/____D/____

e. What was the date of first onset of symptoms for this cancer? ____M/____D/____

14) Has a physician, nurse practitioner, or other health professional ever told y
that (index child) had any other serious disease? If yes, explain:

_____

_____

_____

I.D. #_____

BIRTH DEFECTS

Now, I'd like to ask you about any birth defects that (Index Child) might have.

Was (Index Child) ever diagnosed with any of the following birth defects?

15) Downs' syndrome, also known as mongolism, which is a defect associated with the presence of an extra chromosome in the cells?

1-Yes 8-Refused to answer
2-No 9-Unknown

16) Blooms' syndrome, which is a hereditary disease resulting in stunted growth and skin disorders, such as, enlarged blood vessels and poor skin color?

1-Yes 8-Refused to answer
2-No 9-Unknown

17) Klinefelters' syndrome, which is an inherited disease in males associated with sterility, enlarged breasts and abnormal chromosome patterns in the cells?

1-Yes 8-Refused to answer
2-No 9-Unknown

18) X-linked agammaglobulinemia (or Bruton's Syndrome), which is a reduction of the amount of antibodies in the blood?

1-Yes 8-Refused to answer
2-No 9-Unknown

19) Ataxia telangiectasia, which is a hereditary disease resulting in enlargement of blood vessels in parts of the body, such as, the nose, throat or intestine?

1-Yes 8-Refused to answer
2-No 9-Unknown

20) Wiskott-Aldrich syndrome, which is an inherited disease resulting in chronic e infections, eczema and anemia?

1-Yes 8-Refused to answer
2-No 9-Unknown

IF NO: GO TO NEXT PAGE:

IF YES TO ANY OF THE ABOVE BIRTH DEFECTS;

21) What was the date of diagnosis?_____

MEDICAL PROCEDURES

22a) Did (Index Child) receive any transfusions prior to _____?
[DATE OF DIAGNOSIS]

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT QUESTION

IF YES:	b. What was the date of the transfusion?	c. What was the reason for the transfusion?
	_____M/ _____Y	_____
	_____M/ _____Y	_____
	_____M/ _____Y	_____

23a) Has (Index Child) ever had a tonsillectomy?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT QUESTION

IF YES: b. What was the date of the procedure? _____

24a) Did (Index Child) ever receive x-ray or radiation therapy? (THIS MEANS EXCLUDING DIAGNOSTIC)

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT QUESTION

IF YES, indicate:

b. What was the reason for this therapy?_____

c. What part of the body received the therapy?_____

d. Between which dates was the therapy given? _____M/_____Y _____M/_____Y

e. How many treatments were given?_____

I.D. #_____

Has (Index Child) ever had any of the following immunizations?

IMMUNIZATION	a. EXPOSURE 1-YES 8-REFUSED TO ANSWER 2-NO 9-UNKNOWN	b. WHEN WAS THIS IMMUNIZATION GIVEN?
25) DPT (Diphtheria, pertussis, tetanus)	_____	_____
26) DT (Diphtheria, tetanus)	_____	_____
27) Mumps	_____	_____
28) Rubella	_____	_____
29) Small Pox	_____	_____
30) Measles	_____	_____
31) Polio Vaccine (Oral/Sabin) (INGESTED)	_____	_____
32) Polio Vaccine (Live/Salk) (INJECTED)	_____	_____
33) Other (Indicate):	_____	_____

I.D. #_____

SECTION B: MATERNAL PREGNANCY HISTORY

Now I would like to ask you a series of questions involving your pregnancy history.

1) How many times, in total, have you been pregnant?_____

2) Did your (first/second/etc.) pregnancy result in a

	a. PREG #01	b. PREG #02
1. Live birth?	1	1
2. Stillbirth?	2	2
3. Miscarriage?	3	3
4. Abortion?	4	4
5. Tubal or ectopic pregnancy?	5	5
8. Refused to answer	8	8
9. Unknown	9	9

3) What was the date of the _____? [FILL IN PREVIOUS RESPONSE]

____/____ ____/____
MO YR MO YR

4) How many weeks or months did that pregnancy last? [CIRCLE UNIT]

_____Months _____Months

_____Weeks _____Weeks

IF THE PREGNANCY WAS NOT A LIVE BIRTH, SKIP TO THE NEXT PREGNANCY

5) Was this child male or female?			
	Male	1	1
	Female	2	2
	Refused to answer	8	8
	Unknown	9	9

6) What was (his/her) birthweight?

___ ___ ___ ___
LB OZ LB OZ

c. PREG #03	d. PREG #04	e. PREG #05	f. PREG #06	g. PREG #07
1	1	1	1	1
2	2	2	2	2
3	3	3	3	3
4	4	4	4	4
5	5	5	5	5
8	8	8	8	8
9	9	9	9	9
____/____ MO YR	____/____ MO YR	____/____ MO YR	____/____ MO YR	____/____ MO YR
_____Months	_____Months	_____Months	_____Months	_____Months
_____Weeks	_____Weeks	_____Weeks	_____Weeks	_____Weeks
1	1	1	1	1
2	2	2	2	2
8	8	8	8	8
9	9	9	9	9
___ ___ LB OZ	___ ___ LB OZ	___ ___ LB OZ	___ ___ LB OZ	___ ___ LB OZ

NOTE THE NUMBER OF LIVEBIRTHS HERE: _____

I.D. #_____

BIRTH DEFECTS

IF THE MOTHER HAD MORE THAN ONE LIVE BIRTH, (INDEX CHILD), ASK THE NEXT QUESTI

IF NO, SKIP TO PAGE 29

7a) Were any of (Index Child's brothers or sisters) diagnosed with birth defects?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO PAGE 29

IF YES TO ANY BIRTH DEFECTS:

	1. SIBLING #1	2. SIBLING #2
b. What is the sex of the child?	_____	_____
c. What was the date of birth?	_____	_____
d. What was the birth defect?	_____	_____
e. What was the date of diagnosis?	_____	_____
f. What was the hospital of diagnosis?	_____	_____

[IF HOSPITAL IS UNKNOWN, PROBE BY ASKING FOR THE PHYSICIAN'S NAME AND ADDRESS IF KNOWN]

3. SIBLING #3	4. SIBLING #4	5. SIBLING #5

Now, I am going to ask you some specific questions about your pregnancy with (Index Child).

Were you working outside of your home in:

8) the 3 months before your pregnancy? 1-Yes 2-No 8-Refused to answer 9-Unknown

9) the first 3 months of your pregnancy? 1-Yes 2-No 8-Refused to answer 9-Unknown

10) the second 3 months of your pregnancy? 1-Yes 2-No 8-Refused to answer 9-Unknown

11) the last 3 months of your pregnancy? 1-Yes 2-No 8-Refused to answer 9-Unknown

Were you using any of the following contraceptives in the 3 months before your pregnancy?

12) Birth control pills 1-Yes 2-No 8-Refused to answer 9-Unknown

13) Contraceptive foam 1-Yes 2-No 8-Refused to answer 9-Unknown

14) Jelly, cream, or suppositories alone 1-Yes 2-No 8-Refused to answer 9-Unknown

15) Diaphragm with jelly or cream 1-Yes 2-No 8-Refused to answer 9-Unknown

16) Douche 1-Yes 2-No 8-Refused to answer 9-Unknown

17) IUD, coil or loop 1-Yes 2-No 8-Refused to answer 9-Unknown

18a) Did you use any of the above mentioned contraceptives after the pregnancy had begun?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO SKIP TO THE NEXT QUESTION

IF YES: b. Which contraceptive? _____

c. How long during the preganacy

did you use it?_____

19) Did you receive care from a doctor or other health professional during your pregnancy?

1-Yes 8-Refused to answer
2-No 9-Unknown

IF NO: GO TO QUESTION 22a

IF YES:

20) In which month of your pregnancy did this care begin? _____

21) How many health care visits did you have during your pregnancy in total?

_____

22a) Did you smoke during pregnancy with (Index Child)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO THE NEXT QUESTION

IF YES:

b. During which months of the pregnancy did you smoke?_____

c. During this time about how many cigarettes did you smoke a day?_____

I.D. #_____

23a) During the pregnancy with (INDEX CHILD) did you drink household tap water and/or beverages made from your household tap water such as coffee or juice?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: SKIP TO 23d

IF YES: b) How much coffee or tea did you drink during this pregnancy?

Glasses per day _____

c) How much tap water and beverages made from tap water did you drink per day during the pregnancy?

Glasses per day _____

23d) Did you drink any wine, beer or liquor during pregnancy with (Index Child)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO THE NEXT PAGE

IF YES:

d. During which months of the pregnancy did you drink?_____

During this time,:

	Current (or past) Time			
e. How much wine did you drink a day or week?	|__|	|__|	per	|__|
f. How much beer did you drink a day or week?	|__|__|	|__|	per	|__|
g. How much liquor did you drink a day or week?	|__|__|	|__|	per	|__|

G=glasses C=cans B=bottles O=ounces

D=day W=week M=month

I.D. #_____

Now, I would like to ask about illnesses or health problems you may have experienced during this pregnancy.

24) During this pregnancy, did you ever have:	1. 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	2. What was the date the illness began? (If date unknown try to identify month of pregnancy)
a. measles?	____	_____
b. rubella or German measles?	____	_____
c. chicken pox or shingles?	____	_____
d. influenza or flu?	____	_____
e. hepatitis?	____	_____
f. genital herpes?	____	_____
g. fever for more than 3 days in a row?	____	_____
h. vaginal infections?	____	_____
i. mononucleosis?	____	_____
j. vaginal or uterine bleeding?	____	_____
k. high blood pressure?	____	_____
l. pre-eclampsia or toxemia?	____	_____
m. protein in your urine?	____	_____
n. a urinary tract infection?	____	_____
o. nausea and vomiting for over 3 months?	____	_____
p. cytomegalovirus infection, also called salivary gland virus or inclusion disease?	____	_____
q. aplastic anemia, an anemia due to bone marrow defects?	____	_____
r. any other illnesses or complications I haven't mentioned?	____	_____

Now, I would like to ask you about drugs and medicines you may have taken around the time of your pregnancy with (Index Child). It is important to tell me about medications you received as ointments or injections as well as drugs you took by mouth.

First, I need to know if you breastfed (Index Child) to determine whether I will need to ask about that period.

25) Did you breastfeed (Index Child)? 1-Yes 8-Refused to answer 2-No 9-Unknown

[IF BREASTFED, ASK ABOUT THAT PERIOD. SHOW MOTHER THE MEDICATION LIS

26) In the three months prior to pregnancy or during pregnancy (or while breastfeeding), did you ever take:

	1. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	2. What were the dat of consumption? FROM	TO
a. Amphetamines, such as, Benzedrine, used to decrease nasal congestion, decrease appetite and as a stimulant for depression?	____	____M/____Y	____M/
b. Chloramphenicol, used in treating resistant infections?	____	____M/____Y	____M/
c. Chlorpromazine, used to treat nausea or calm nerves?	____	____M/____Y	____M/
d. Griseofulvin, used to treat fungus infections?	____	____M/____Y	____M/
e. Oxyphenbutazone, used to treat inflammation which causes pain and cannot be controlled with aspirin?	____	____M/____Y	____M
f. Phenylbutazone, such as, Butazolidin^{R}, used to treat inflammation which causes pain and cannot be controlled with aspirin?	____	____M/____Y	____
g. Propylthiouracil, used to treat an overactive thyroid?	____	____M/____Y	____
h. Tolbutamide, also known as, Orinase^{R}, used to treat diabetes?	____	____M/____Y	____

1. EXPOSURE	2. What were the dates of consumption? FROM	TO

i. Any other type of prescribed medication that I haven't mentioned? (SPECIFY)

1-Yes 8-Refused to answer
2-No 9-Unknown

_____	___M/___Y	___M/___Y
_____	___M/___Y	___M/___Y
_____	___M/___Y	___M/___Y

Now, I would like to ask about non-prescription drugs. You may consider these questions to be sensitive, but I would like to reassure you that this information will be kept completely confidential.

j. In the 3 months prior to pregnancy or during the pregnancy (or while breastfeeding), did you ever use recreational drugs such as marijuana, cocaine, or LSD?

1-Yes 8-Refused to answer ___M/___Y ___M/___Y
2-No 9-Unknown

Now, I will ask you about diagnostic procedures such as x-rays which you might have received during this pregnancy.

[CIRCLE X-RAY NUMBER FOR EACH PROCEDURE REPORTED, NOTE PROCEDURE NUMBER]

27) In the 3 months prior to your pregnancy or during your pregnancy, did you ever have any of the following diagnostic procedures?

	1. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	2. What was/were the date(s) of occurrence?
a. amniocentesis?	___	___M/___Y
b. ultrasound?	___	___M/___Y
c. upper GI series?	___	___M/___Y

I.D. #_____

	1. EXPOSURE 1-YES 8-REFUSED TO ANSWER 2-NO 9-UNKNOWN	2. What was/were the date(s) of occurrence?
d. x-ray of the gallbladder?	____	____M/____Y
e. barium enema?	____	____M/____Y
f. IVP or cystogram?	____	____M/____Y
g. other x-rays of lower abdomen? [SPECIFY]_____	____	____M/____Y
h. x-ray of lower back?	____	____M/____Y
i. CAT scan?	____	____M/____Y
j. x-ray of baby's size and position?	____	____M/____Y
k. radiation therapy?	____	____M/____Y
l. X-ray of abdomen?	____	____M/____Y

	1. In the three months prior to your pregnancy or during your pregnancy, did you ever have any of the following diagnostic procedures?	2. Were you usually shielded from the x-ray with a lead apron over your abdomen?	3. What was/ were the dates of occurrence?
	1-YES 8-REFUSED TO ANSWER 2-NO 9-UNKNOWN		
m. chest X-ray?	____	____	____M/____Y
n. dental therapy?	____	____	____M/____Y
o. other x-rays such as for injuries? [SPECIFY]_____	____	____	____M/____Y

Now, I would like to ask about anesthetics, including general anesthetics to put you to sleep during surgery, or local anesthetics such as novacaine for dental procedures.

28a) Did you receive any anesthetic at any time in the 3 months prior to your pregnancy or during the pregnancy (or while breastfeeding)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT PAGE

IF YES:

		ANESTHETIC #1	ANESTHETIC #2
b. Was it a general anesthetic to put you to sleep or a local anesthetic to reduce pain?	1. General	1	1
	2. Local	2	2
	8. Refused to answer	8	8
	9. Unknown	9	9
c. For what reason did you receive the anesthetic? [DON'T READ LIST]	1. Surgery	1	1
	2. Dental	2	2
	3. Other [SPECIFY]_____(_)	_____(_)	_____(_)
	8. Refused to answer	8	8
	9. Unknown	9	9
d. What was the date the anesthetic was used?		____M/____Y	____M/____Y

e. Did you received any other anesthetics in this period?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO NEXT PAGE

IF YES: f. What was the reason for the anesthetic?

_____

_____

I.D. #_____

In the 3 months prior to your pregnancy or during your pregnancy did you have exposure to:

a. b. At what time during your pregnancy were you exposed?

29) Pesticides, like roach or garden spray?

1-Yes 8-Refused to answer IF YES: ____M/____Y
2-No 9-Unknown

____M/____Y

30) Herbicides, like weedkiller?

1-Yes 8-Refused to answer IF YES: ____M/____Y
2-No 9-Unknown

____M/____Y

31) Oven cleaner?

1-Yes 8-Refused to answer IF YES: ____M/____Y
2-No 9-Unknown

____M/____Y

32) A heating pad or electric blanket on a regular basis?

1-Yes 8-Refused to answer IF YES: ____M/____Y
2-No 9-Unknown

____M/____Y

Now, I want to ask specifically about cancer in (Index Child's) family. Leukemia, Lymphoma, and Hodgkins Disease are all types of cancer and should be mentioned, along with other types of cancer if they occurred.

33) First, I would like you to take your time and try to focus on each of (Index Child's) following relatives and try to recall if they were ever diagnosed with cancer.

	1. 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	2. What was the type of cancer?	3. What was the date of diagnosis?
(INDEX CHILD'S):			
a. Maternal grandmother	____	_____	_____
b. Maternal grandfather	____	_____	_____
c. Paternal grandmother	____	_____	_____
d. Paternal grandfather	____	_____	_____
e. Mother	____	_____	_____
f. Father	____	_____	_____
g. Sister(s)	____	_____	_____
h. Brother(s)	____	_____	_____

I.D. #_____

PART V. EXPOSURE DATA

SECTION A: ENVIRONMENTAL EXPOSURE

1) Now, I would like to ask you about school(s) that (Index Child) attended. Please begin with the first school, including preschool, that (Index Child) attended.

	1. What was the name of the first (next) school (Index Child) attended?	2. During which dates did he/she attend school there?	
	NAME OF SCHOOL/TOWN	DATES FROM	TO
a.	_____	____M/____Y	____M/____Y
b.	_____	____M/____Y	____M/____Y
c.	_____	____M/____Y	____M/____Y
d.	_____	____M/____Y	____M/____Y
e.	_____	____M/____Y	____M/____Y
f.	_____	____M/____Y	____M/____Y
g.	_____	____M/____Y	____M/____Y
h.	_____	____M/____Y	____M/____Y

Now, I would like to ask you some questions regarding (Index Child's) hobbies and activities.

2) Did (Index Child) eat fruit or vegetables raised in the family garden or a neighbor's garden?

1-Yes 2-No 8-Refused to answer 9-Unknown

3) Did (Index Child) go fishing in lakes and streams in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

4a) Did (Index Child) eat fish caught in the lakes and streams in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO THE NEXT QUESTION

IF YES:

b. What is the name of the body of water where the fish were caught?_____

5a) Did (Index Child) regularly swim in lakes and rivers in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: SKIP TO NEXT QUESTION

IF YES: b. What are the names of these bodies of water?

_____

6a) Did (Index Child) regularly participate in any activities or hobbies which would bring him/her into contact with chemicals, fumes, dust or radiation?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT PAGE

IF YES: b. What types of activities were these?

Describe:_____

7a) Did (Index Child) regularly attend a specific church or temple?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT QUESTION

IF YES: b. What is its name?_____

c. In which city or town is it located?_____

8a) Did (Index Child) actively belong to any community organizations (e.g., Boyscouts, Little League, etc.)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT QUESTION

IF YES: b. Which was this?_____

c. Where did they meet regularly?_____

9a) Did (Index Child) regularly travel away from home (e.g., summer camp, regular vacation, etc.)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT PAGE

IF YES: b. What was the name and location of this destination?

_____

I.D. #_____

SECTION B: HOME EXPOSURES

Now, I would like to ask about some electrical appliances that (Index Child) may have used before or during 19_ _ [DATE OF DIAGNOSIS OF CASE].

Did (he/she) regularly use (a/an):

1) Electric blanket? 1. Yes 2. No 8. Refused to answer 9. Unknown

2) Heated water bed? 1. Yes 2. No 8. Refused to answer 9. Unknown

3) Heating pad? 1. Yes 2. No 8. Refused to answer 9. Unknown

IF NO TO ALL SECTIONS OF THE ABOVE QUESTION, SKIP TO NEXT PAGE

IF YES TO ANY PART, CONTINUE BELOW WITH APPROPRIATE QUESTIONS ONLY

Over what ages did (he/she) use the:

4) Electric blanket? Age:_____years to _____years

5) Heated water bed? Age:_____years to _____years

6) Heating pad? Age:_____years to _____years

About how many months out of the year did (he/she) use the:

7) Electric blanket? __ __Months

8) Heated water bed? __ __Months

9) Heating pad? __ __Months

SECTION C: ANIMAL EXPOSURES

Now, I would like to ask you some questions about farm animals and pets (Index Child) may have been in contact with.

We'll first begin with farm animals.

1) Did (Index Child) ever live on a farm where animals were kept?

1. Yes
2. No
8. Refused to answer
9. Unknown

IF NO: GO TO THE NEXT PAGE

IF YES:

	a. ANIMAL #01	b. ANIMAL #02	c. ANIMAL #03	d, ANIMAL #04
2) What types of animals were on the farm?	_____	_____	_____	_____
3) Over what years was (he/she) on the farm with these animals	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _
4) Did any of the farm animals have leukemia?				
Yes	1	1	1	1
No	2	2	2	2
Ref	8	8	8	8
Unk	9	9	9	9
5) What year did the animal have leukemia?	19_ _	19_ _	19_ _	19_ _

Now, I'll ask you some questions about household pets.

6) Did (Index Child) ever have any cats?

1. Yes
2. No
8. Refused to answer
9. Unknown

IF NO: GO TO QUESTION 10

IF YES:	a. CAT #01	b. CAT #02	c. CAT #03	d. CAT #04
7) Over what years did he/she have this cat?	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ to 19_

8) Did (this cat/any of these cats) ever have leukemia or cancer?

Yes	1	1	1	1
No	2	2	2	2
Refused	8	8	8	8
Unk	9	9	9	9

IF NO: GO TO THE NEXT QUESTION

IF YES:

9) What year was/were the cat(s) diagnosed with leukemia or cancer?	19_ _	19_ _	19_ _	19_

[IF INDEX CHILD'S FATHER DOES NOT RESIDE IN THE SAME HOUSEHOLD AS THE INDEX CHILD'S MOTHER, THEN ASK THE STATEMENT BELOW. OTHERWISE SKIP TO PAGE 47, QUESTION 11.]

10a) I have some questions concerning (index child's) father. Would it be alright with you if I contacted him?

If Yes: b. Do you know his address and phone number?

_____

_____

_____

GO TO QUESTION 12

If No: c. Do you mind then answering a few questions as best you can for h

If yes: GO TO FATHER'S QUESTIONNAIRE.

If no: GO TO PAGE 47, QUESTION 12.

e. CAT #05	f. CAT #06	g. CAT #07	h. CAT #08
_____	_____	_____	_____
From 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _
YES.....1	1	1	1
NO.....2	2	2	2
REFUSED..8	8	8	8
UNKNOWN..9	9	9	9
19_ _	19_ _	19_ _	19_ _

11) IF FATHER AND MOTHER WERE INTERVIEWED SIMULTANEOUSLY, GO TO FATHER'S QUESTIONNAIRE NOW AND COMPLETE APPROPRIATE PART.

IF FATHER AND MOTHER ARE TO BE INTERVIEWED SEPARATELY IN THE SAME HOUSEHOLD, READ: Is Mr._____ available to complete his portion of the interview now? [GO TO FATHER'S QUESTIONNAIRE]

12) This concludes the childhood leukemia health study questionnaire.

Do you have any personal thoughts or concerns you wish to share regarding the environment and how it may be affecting the health of Woburn's citizens?

_____

_____

_____

_____

_____

_____

_____

IF CHILD IS A CONTROL, GO TO THE NEXT PAGE, LAST PARAGRAPH.

IF CHILD IS A CASE AND PRESENTLY >= EIGHTEEN YEARS OF AGE, SKIP TO NEXT QUESTION, ELSE READ:

It may also be necessary for us to consult your child's medical records to help prop interpret and expand upon the data you have given me. Could you please read and sig this authorization for disclosure of medical information.
[GO TO NEXT PAGE, LAST PARAGRAPH]

Could you please provide me with your child's full name, current address and telephone number.

CASE'S NAME: _____

CURRENT ADDRESS: _____

_____

TELEPHONE NUMBER: _____

We want to thank you very much for participating in this health study. Your cooperation is essential for us to learn more about the causes of disease and eventually ways of preventing disease. I want to reassure you that all the information you provided will be kept confidential.

INDEX CHILD'S STUDY NUMBER _____

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
THE DIVISION OF ENVIRONMENTAL
EPIDEMIOLOGY AND TOXICOLOGY

WOBURN CHILDHOOD HEALTH STUDY QUESTIONNAIRE

FATHER'S QUESTIONNAIRE

INDEX CHILD'S STUDY NUMBER: _____

Sex of (Index Child) 1-Male 2-Feme

PART I: INTRODUCTION

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
THE DIVISION OF ENVIRONMENTAL
EPIDEMIOLOGY AND TOXICOLOGY

WOBURN CHILDHOOD HEALTH STUDY QUESTIONNAIRE

1) INDEX CHILD'S NAME AND ADDRESS: _____

_____

_____

2) INTERVIEWEE'S NAME AND ADDRESS: _____

_____

_____

3) INTERVIEWEE'S TELEPHONE #: _____

4) RELATIONSHIP OF INTERVIEWEE TO INDEX CHILD: _____

5) DATE OF INTERVIEW: _____

"Hello, my name is _____. I am representing the Massachusetts Department of Public Health and am here with regard to our appointment for the completion of the Woburn Childhood Health Study Questionnaire. May I come in?"

"As you may be aware, this study is being conducted among a sample of children chosen at random from the general population. Your child has been one of those selected to be included in this study. Questions will be mainly about your work history, where you have lived, and you and your child's medical histories and hobbies. Did you have a chance to review the letter I sent you describing this study?"

I.D. #_____

F YES: "Do you have any specific questions?"

"Let me remind you that your cooperation is completely voluntary. You have the right to withdraw from the interview at any time. All information collected in this study will be locked and kept in the strictest confidence in accordance with Massachusetts General Laws. No use will be made of information that would identify you to anyone outside this project."

IF NO: "Let me read it to you." [READ LETTER]

Before we begin, could you please sign this consent form which indicates that you understand the purpose of this study and your rights as a participant as I have just reviewed them with you."

"Feel free to take as much time as you need to answer the questions. Some of them concern events which took place many years ago and we would like you to answer them as completely and carefully as you can. If there are any questions you do not fully understand, please let me know. If you are unsure of an answer to as specific question, please say so."

I.D. #_____

PART II: MILITARY HISTORY

First let me ask you about your military service history.

1a) Did you serve in the Armed Forces prior to (index child's mother's) pregnancy with (Index Child)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT PAGE

IF YES:

b. Which branch did you serve in? _____

c. What were your dates of service?_____

d. Did you serve in Vietnam, Laos, or Cambodia?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT PAGE

IF YES:

e. Where did you serve?	f. What were your dates of service?
_____	_____
_____	_____
_____	_____
_____	_____

I.D. #_____

PART III: OCCUPATIONAL INFORMATION

SECTION A: OCCUPATIONAL HISTORY

Now, I have some questions about your occupational history starting with the job you held during _____ [MONTH AND YEAR OF DIAGNOSIS OF CASE] back to _____ [TWO YEARS PRIOR TO CONCEPTION OF INDEX CHILD].

We are looking for employer, job title, nature of the work, duration and potential exposures. Please be sure to note if there were any job title or type of work changes within the same company. [NOTE: PROMPT THIS AT THE END OF EACH JOB DESCRIPTION]

IDENTIFICATION NUMBER _____

OCCUPATIONAL HISTORY

JOB #	a. WHAT IS/WAS YOUR (___'S) EMPLOYER'S NAME & ADDRESS?	b. WHAT KIND OF BUSINESS WAS IT?	c. WHAT WAS THE TITLE OR NAME OF THAT JOB?	d. WAS T JOB F OR PA TIM
1	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL.. PART..
2	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL.. PART..
3	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL. PART.
4	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL. PART.
5				FULL PART

JOB #	a. WHAT IS/WAS YOUR (____'S) EMPLOYER'S NAME & ADDRESS?	b. WHAT KIND OF BUSINESS WAS IT?	c. WHAT WAS THE TITLE OR NAME OF THAT JOB?	d. WAS TH JOB FULL OR PART TIME?
6	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL...1 PART...2
7	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL...1 PART...2
8	UNKNOWN...9	UNKNOWN...9	UNKNOWN...9	FULL...1 PART...2

IDENTIFICATION NUMBER _____

OCCUPATIONAL HISTORY CONTINUED

JOB #	e. WHAT WAS THE NATURE OR TYPE OF WORK YOU DID?	f. WHAT MONTH & YEAR DID THIS JOB START?	g. WHAT MONTH & YEAR DID THIS JOB END?	h. DID YOU DRINK BOTTLED WATER AT THIS JOB?	i. DURING THAT JOB DID YOU USE OR WERE YOU EXPOSED TO: (ask individually)
1		____/____ MO YR	____/____ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK --- -- --- CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
2		____/____ MO YR	____/____ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK --- -- --- CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
3		____/____ MO YR	____/____ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK --- -- --- CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
4		____/____ MO YR	____/____ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK --- -- --- CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
5		____/____ MO YR	____/____ MO YR	1 Yes 2 No 8 Refused 9 Unknown	YES NO UNK --- -- --- CHEMICALS 1 2 9 FUMES 1 2 9 RADIATION 1 2 9 IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		

JOB #	e. WHAT WAS THE NATURE OR TYPE OF WORK YOU DID?	f. WHAT MONTH & YEAR DID THIS JOB START?	g. WHAT MONTH & YEAR DID THIS JOB END?	h. DID YOU DRINK BOTTLED WATER AT THIS JOB?	i. DURING THAT JOB DID YOU USE OR WERE YOU EXPOSED TO: (ask individually)
				1 Yes	YES NO UNK
				2 No	--- -- ---
				8 Refused	CHEMICALS 1 2 9
6		___/___	___/___	9 Unknown	FUMES 1 2 9
		MO YR	MO YR		RADIATION 1 2 9
					IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
				1 Yes	YES NO UNK
				2 No	--- -- ---
				8 Refused	CHEMICALS 1 2 9
7		___/___	___/___	9 Unknown	FUMES 1 2 9
		MO YR	MO YR		RADIATION 1 2 9
					IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		
				1 Yes	YES NO UNK
				2 No	--- -- ---
				8 Refused	CHEMICALS 1 2 9
8		___/___	___/___	9 Unknown	FUMES 1 2 9
		MO YR	MO YR		RADIATION 1 2 9
					IF YES, SPECIFY:
	UNKNOWN...9	UNK...9	UNK...9		

I.D. #_____

SECTION B: EXPOSURE HISTORY

I am now going to read you a list of chemicals. [SHOW LIST TO PARENT] Please tell me if you can remember ever using or being exposed to any of these chemicals in any of your jobs. If you are unsure, please say so. Were you exposed to (READ LIST):

CHEMICAL NAME	a. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	b. DURING WHICH DATES WERE YOU EXPOSED? FROM M/ Y TO M/ Y	c. AT WHICH JOBS WERE YOU EXPOSED TO THIS CHEMICAL?	d. HOW OFTEN WERE YOU EXPOSED? 1-ALWAYS 2-SOMETIMES 3-RARELY
1) COKE OVEN EMISSIONS	____	M/ Y M/ Y	_____	_____
2) LEAD	____	M/ Y M/ Y	_____	_____
3) POLYVINYL CHLORIDE	____	M/ Y M/ Y	_____	_____
4) CHLOROFORM	____	M/ Y M/ Y	_____	_____
5) IONIZING RADIATION	____	M/ Y M/ Y	_____	_____
6) PESTICIDES	____	M/ Y M/ Y	_____	_____
7) HERBICIDES	____	M/ Y M/ Y	_____	_____
8) SOLVENTS	____	M/ Y M/ Y	_____	_____

I.D. #_____

Did you ever use or were you ever exposed to any of these specific chemicals, while at work?

CHEMICAL NAME	a. EXPOSURE 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	b. DURING WHICH DATES WERE YOU EXPOSED? FROM M/ Y	TO M/ Y	c. AT WHICH JOB WERE YOU EXPOSED TO THIS CHEMICAL?	d. HOW OFTEN WERE YOU EXPOSED? 1-ALWAYS 2-SOMETIMES 3-RARELY
9) CARBONTETRACHLORIDE	____	M/ Y	M/ Y	_____	_____
10) BENZENE	____	M/ Y	M/ Y	_____	_____
11) TOLUENE	____	M/ Y	M/ Y	_____	_____
12) XYLENE	____	M/ Y	M/ Y	_____	_____
13) TRICHLOROETHYLENE	____	M/ Y	M/ Y	_____	_____
14) METHYLENE CHLORIDE	____	M/ Y	M/ Y	_____	_____

VIEWER:
OMPLETE THE REMAINDER OF THE QUESTIONNAIRE WITH THE FATHER IF HE WILL BE
NTERVIEWED SEPARATELY EITHER IN THE SAME HOUSE OR AT A DIFFERENT ADDRESS.
LSE RETURN TO THE MOTHERS QUESTIONNAIRE PAGE 47, #12.

HE RESIDENTIAL HISTORY SECTION WHICH FOLLOWS, PERTAINS ONLY TO THE TIME PERIOD WHEN
HE FATHER AND CHILD LIVED IN THE SAME HOUSEHOLD.

PART IV: RESIDENTIAL HISTORY

Now, I would like to ask you about residences you and (Index Child) have lived in. We will begin with the residence you lived in during _____ [MONTH AND YEAR OF DIAGNOSIS OF CASE] and work back to the residence you lived in during _____ [MONTH AND YEAR 2 YEARS PRIOR TO CONCEPTION OF INDEX CHILD].

[IF INDEX CHILD IS ADOPTED OR LIVED WITH A GUARDIAN, THEN ASK FOR THE RESIDENCES THE CHILD MAY HAVE LIVED IN PRIOR TO LIVING WITH INTERVIEWEE]

IDENTIFICATION NUMB_____

RESIDENTIAL HISTORY

	a. WHAT WAS THE ADDRESS OF THE _____ (1ST, 2ND, ETC.) RESIDENCE?	b. FROM WHAT YEAR TO WHAT YEAR WAS THAT?	c. HOW WAS THE WATER SUPPLIED TO THIS RESIDENCE?	d. WHILE LIVING IN THIS HOUSEHOLD DID (INDEX CHILD) DRINK MOSTLY...
SIO.				
).				
1		_____ to _____	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
	9 UNKNOWN	9-UNKNOWN		
2		_____ to _____	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
	9 UNKNOWN	9-UNKNOWN		
3		_____ to _____	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
	9 UNKNOWN	9-UNKNOWN		
4		_____ to _____	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused 9 Unknown
	9 UNKNOWN	9-UNKNOWN		
5		_____ to _____	1 Private Well 2 Public Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 8 Refused

IDENTIFICATION NUMBER _____

RESIDENTIAL HISTORY CONTINUED

e.	f.
HOW MANY PEOPLE SMOKED CIGARETTES, CIGARS OR PIPES IN THAT HOUSEHOLD DAILY?	DID THIS RESIDENCE HAVE ANY OF THE FOLLOWING FEATURES? PLEASE RESPOND AFTER EACH ITEM.
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garag 8 Refused 9 Unknown 7.00
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown

IDENTIFICATION NUMBER_____

RESIDENTIAL HISTORY

RESID. NO.	a. WHAT WAS THE ADDRESS OF THE _____ (1ST, 2ND, ETC.) RESIDENCE?	b. FROM WHAT YEAR TO WHAT YEAR WAS THAT?	c. HOW WAS THE WATER SUPPLIED TO THIS RESIDENCE?	d. WHILE LIVING IN THIS HOUSEHOLD DID (INDEX CHILD) DRINK MOSTLY...
6	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
7	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
8	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
9	9 UNKNOWN	_____ to _____ 9-UNKNOWN	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused 5 Unknown
10		_____ to _____	1 Private Well 2 Public Water 3 Bottled Water 8 Refused 9 Unknown	1 Household tap water and beverages made from tap water, i.e. coffee or juice 2 Bottled water and beverages made from bottled water such as coffee or juice 3 Store-bought beverages, i.e. soda, beer, milk, or juice not needing added water 4 Refused

IDENTIFICATION NUMBER _____

RESIDENTIAL HISTORY CONTINUED

e. HOW MANY PEOPLE SMOKED CIGARETTES, CIGARS OR PIPES IN THAT HOUSEHOLD DAILY?	f. DID THIS RESIDENCE HAVE ANY OF THE FOLLOWING FEATURES? PLEASE RESPOND AFTER EACH ITEM.
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds. How many times in total?_____ ___use of "no-pest" strips ___attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds. How many times in total?_____ ___use of "no-pest" strips attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds. How many times in total?_____ ___use of "no-pest" strips attached garage 8 Refused 9 Unknown
# of smokers_____ 8 Refused 9 Unknown	___nearby tree or agricultural spraying How many times in total?_____ ___pest or insect extermination in house How many times in total?_____ ___mosquito spraying or fogging How many times in total?_____ ___insecticide or herbicide treatment of yard to control insects or weeds. How many times in total?_____ ___use of "no-pest" strips attached garage 8 Refused 9 Unknown

I.D. #_____

PART V: MEDICAL HISTORY

Now, I want to ask specifically about cancer in (Index Child's) family. Leukemia, Lymphoma, and Hodgkins Disease are all types of cancer and should be mentioned, along with other types of cancer if they occurred.

1) First, I would like you to take your time and try to focus on each of (Index Child's) following relatives and try to recall if they were ever diagnosed with cancer.

	1. 1-YES 2-NO 8-REFUSED TO ANSWER 9-UNKNOWN	2. What was the type of cancer?	3. What was the date of diagnosis?
(INDEX CHILD'S):			
a. Maternal grandmother	____	_____	_____
b. Maternal grandfather	____	_____	_____
c. Paternal grandmother	____	_____	_____
d. Paternal grandifather	____	_____	_____
e. Mother	____	_____	_____
f. Father	____	_____	_____
g. Sister(s)	____	_____	_____
h. Brother(s)	____	_____	_____

I.D. #_____

PART VI. EXPOSURE DATA

SECTION A: ENVIRONMENTAL EXPOSURE

1) Now, I would like to ask you about school(s) that (Index Child) attended. Please begin with the first school, including preschool, that (Index Child) attended.

	1. What was the name of the first (next) school (Index Child) attended?	2. During which dates did he/she attend school there?	
	NAME OF SCHOOL/TOWN	DATES FROM	TO
a.	_____	____M/____Y	____M/____Y
b.	_____	____M/____Y	____M/____Y
c.	_____	____M/____Y	____M/____Y
d.	_____	____M/____Y	____M/____Y
e.	_____	____M/____Y	____M/____Y
f.	_____	____M/____Y	____M/____Y
g.	_____	____M/____Y	____M/____Y
h.	_____	____M/____Y	____M/____Y

I.D. #_____

Now, I would like to ask you some questions regarding (Index Child's) hobbies and activities.

2) Did (Index Child) eat fruit or vegetables raised in the family garden or a neighbor's garden?

1-Yes 2-No 8-Refused to answer 9-Unknown

3) Did (Index Child) go fishing in lakes and streams in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

4a) Did (Index Child) eat fish caught in the lakes and streams in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO THE NEXT QUESTION

IF YES:

b. What is the name of the body of water where the fish were caught?_____

5a) Did (Index Child) regularly swim in lakes and rivers in Woburn?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: SKIP TO NEXT QUESTION

IF YES: b. What are the names of these bodies of water?

_____

6a) Did (Index Child) regularly participate in any activities or hobbies which would bring him/her into contact with chemicals, fumes, dust or radiation?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO, SKIP TO NEXT PAGE

IF YES: b. What types of activities were these?

Describe:_____

I.D. #_____

7a) Did (Index Child) regularly attend a specific church or temple?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT QUESTION

IF YES: b. What is its name?_____

c. In which city or town is it located?_____

8a) Did (Index Child) actively belong to any community organizations (e.g., Boyscouts, Little League, etc.)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT QUESTION

IF YES: b. Which was this?_____

c. Where did they meet regularly?_____

9a) Did (Index Child) regularly travel away from home (e.g., summer camp, regular vacation, etc.)?

1-Yes 2-No 8-Refused to answer 9-Unknown

IF NO: GO TO THE NEXT PAGE

IF YES: b. What was the name and location of this destination?

_____

I.D. #_____

SECTION B: HOME EXPOSURES

Now, I would like to ask about some electrical appliances that (Index Child) may have used before or during 19_ _ [DATE OF DIAGNOSIS OF CASE].

Did (he/she) regularly use (a/an):

1) Electric blanket? 1. Yes 2. No 8. Refused to answer 9. Unknown

2) Heated water bed? 1. Yes 2. No 8. Refused to answer 9. Unknown

3) Heating pad? 1. Yes 2. No 8. Refused to answer 9. Unknown

IF NO TO ALL SECTIONS OF THE ABOVE QUESTION, SKIP TO NEXT PAGE

IF YES TO ANY PART, CONTINUE BELOW WITH APPROPRIATE QUESTIONS ONLY

Over what ages did (he/she) use the:

4) Electric blanket? Age:_____years to _____years

5) Heated water bed? Age:_____years to _____years

6) Heating pad? Age:_____years to _____years

About how many months out of the year did (he/she) use the:

7) Electric blanket? __ __Months

8) Heated water bed? __ __Months

9) Heating pad? __ __Months

SECTION C: ANIMAL EXPOSURES

Now, I would like to ask you some questions about farm animals and pets (Index Child) may have been in contact with.

We'll first begin with farm animals.

1) Did (Index Child) ever live on a farm where animals were kept?

1. Yes
2. No
8. Refused to answer
9. Unknown

IF NO: GO TO THE NEXT PAGE

IF YES:

	a. ANIMAL #01	b. ANIMAL #02	c. ANIMAL #03	d. ANIMAL #04
2) What types of animals were on the farm?	_____	_____	_____	_____
3) Over what years was (he/she) on the farm with these animals	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _
4) Did any of the farm animals have leukemia?				
Yes	1	1	1	1
No	2	2	2	2
Ref	8	8	8	8
Unk	9	9	9	9
5) What year did the animal have leukemia?	19_ _	19_ _	19_ _	19_ _

Now, I'll ask you some questions about household pets.

6) Did (Index Child) ever have any cats?

1. Yes 2. No
8. Refused to answer 9. Unknown

IF NO: GO TO QUESTION 10

IF YES:	a. CAT #01	b. CAT #02	c. CAT #03	d. CAT #04
7) Over what years did he/she have this cat?	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _

8) Did (this cat/any of these cats) ever have leukemia or cancer?

Yes	1	1	1	1
No	2	2	2	2
Refused	8	8	8	8
Unk	9	9	9	9

IF NO: GO TO THE NEXT QUESTION

IF YES:

9) What year was/were the cat(s) diagnosed with leukemia or cancer?	19_ _	19_ _	19_ _	19_ _

RETURN TO MOTHER'S QUESTIONNAIRE, PAGE 47, QUESTION 12.

e. CAT #05	f. CAT #06	g. CAT #07	h. CAT #08
_____	_____	_____	_____
From 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _	From 19_ _ to 19_ _
YES.....1	1	1	1
NO.....2	2	2	2
REFUSED..8	8	8	8
DKNOWN..9	9	9	9
19_ _	19_ _	19_ _	19_ _

APPENDIX XI

Murphy Water Distribution Model

SUMMARY

In May of 1979 the Massachusetts Department of Environmental Quality Engineering (DEQE) discovered that Wells G and H, two public drinking water wells in Woburn, Massachusetts, were contaminated with toxic chemicals. These wells were then shut off.

Subsequent studies of the health of the people of Woburn have indicated that the city had a higher level of childhood illness than would normally be expected. Research is currently being undertaken to determine the rate of adverse reproductive outcomes within Woburn, and to test associations between these outcomes and exposure to environmental contaminants, including the water from Wells G and H during the periods of their operation. This report presents the calculation of this exposure as a function both of roughly fifty hydraulically distinct neighborhoods within Woburn and of the 114 months of Wells' G and H operation.

The method used to calculate this exposure to water from the wells begins with a computer model of the water distribution system that was developed by the author under a previous contract with DEQE. This Woburn water distribution model was applied to the various pumping and water use configurations that occurred during each month that Wells G and H were in operation. The results of these calculations were then individually analyzed with a hydraulic mixing model to calculate the mixture of water supplied to each neighborhood. Finally, the resulting mixtures were combined in proportion to the period of their occurence

during each month to provide a monthly average exposure index for each neighborhood and each month. These indices were also summed to determine the cumulative exposure.

The validity and error levels of the distribution and mixing models were analyzed by comparing computer predictions of fluoride concentration distributions in both San Jose, California and Woburn with concentrations measured during field tests. The locations of the boundary between the zones with and without the fluoride tracer were predicted within one pipe junction of where they were observed. The root-mean-square differences between predicted and observed dilutions were roughly thirty percent.

The levels of exposure to water from Wells G and H were found to vary widely as is shown by Figures 10 to 15. Typically the neighborhoods south and west of the center of the city, Main Street and Montvale Avenue, received no or very little water from Wells G and H. The neighborhoods of east Woburn along and near Washington Street received water mostly from Wells G and H whenever those wells were pumping. The mixture zone between the two water sources ran along, or just to the east of, Main Street.

I. Introduction

In May of 1979 the Massachusetts Department of Environmental Quality Engineering discovered that Wells G and H in Woburn, Massachusetts were contaminated with toxic chemicals. These wells were shut off and have not been used for water supply since then, but they had been in service for 2995.5 days since their installation in 1964. The rest of the city's wells were not contaminated and continue in operation. The Metropolitan District Commission supplied the water required to replace the water that had been supplied by the two wells.

Various studies (7,8,9) of the health of the people of Woburn indicate that part of the city had a higher incidence of certain childhood illnesses, including lymphocytic leukemia, than would normally be expected. The affected part of Woburn roughly coincided with the area served by the two contaminated wells. The water distribution system of Woburn was analyzed by Helen A. Waldorf and Robert K. Cleary of the New England Interstate Water Pollution Control Commission. Based on their 1984 report(10), a 1986 journal article(2) by Lagakos, Wessen, and Zelen has reported that the rates of childhood illness have "a statistically significant positive association" with the use of water from the two contaminated wells. A second water distribution analysis(3), made by the author to evaluate and complement the study of Waldorf and Cleary, concurred with their overall assessment.

At present the Centers for Disease Control, the Massachusetts

Department of Public Health and the Massachusetts Health Research Institute are jointly working under a Cooperative Agreement to test statistical correlations between environmental exposure to various chemicals and reproductive outcomes in Woburn. Various media (air, water, soil) in Woburn are known to be contaminated, and so exposure through all these media will be examined as part of this Cooperative Agreement. The water from Wells G and H is one pathway of this exposure. Thus the calculation of the degree of exposure to contaminated water from Wells G and H is needed for this study. The Massachusetts Health Research Institute has contracted with the author for the performance of that work. This report presents the computer model of exposure of exposure to water from Wells G and H.

The ideal description of exposure to water from Wells G and H is the determination of the concentration history of each of the various hazardous chemicals in the water delivered to each residence during the active lifetime of the wells. First, the history of the concentration of each chemical at the two wells must be described. Although hydrogeologic investigations and groundwater quality evaluations(1,6) have determined some characteristics of the groundwater contaminant plumes that supplied water to Wells G and H, the wells' chemical history data are limited. The only measurements of contaminant concentrations of the well water were those leading to the shutdown of the wells. This report does not address this issue, but rather assumes that the wells were contaminated whenever they

were pumping. The second part of the exposure description is the determination of the history of the distribution of the water from Wells G and H to describe the amount of that water used by every residence in Woburn. This report presents a study of G and H exposure using the author's water distribution model to calculate exposure in greater detail than was presented in the authors's previous report(3).

That report, "Water Distribution in Woburn, Massachusetts," only presented flow patterns during twelve characteristic months, with each pattern dividing Woburn into three zones: Zone A with no G and H water, Zone B with G and H water, and Zone C with some mixture of G and H water and water from the other wells. In the present study the author's water distribution models for the 1964-1969 pipe network and the 1970-1979 pipe network are applied as before to obtain water distributions for each month of the exposure period. An additional computer program, which accounts for the mixing of water from Wells G and H and the other Woburn wells during each month, is used to calculate an "exposure index" for over fifty hydraulically different neighborhoods within Woburn. This exposure index is the product of the fraction of the month when any contaminated water reached a particular neighborhood and, during that period of the month, the fraction of the water delivered to that neighborhood which came from the contaminated wells. For example if, during half of June, one third of the water at node 40 came from Wells G and H, then the exposure index at node 40 for June would be one sixth.

Since this report is founded on the author's previous report,that report is referenced often herein. Readers interested in the details of the water distribution model are referred to that report(3).

II. Review of Woburn Water Distribution Model

In general the Woburn water supply system takes water pumped from the ground at the City's wells and delivers it through a pipe network to the residential and business consumers of water. In order to deliver the amounts of water needed by the consumers, the system's operators use pump control valves and water storage tanks and reservoirs to regulate the water distribution. This section of the report describes this water supply system through the use of computer models. Since this study is founded on the author's previous report, "Water Distribution in Woburn, Massachusetts," that report is referenced often herein. Readers interested in the details of the water distribution model are referred to that report (3).

IIa. Woburn Water System Data

During the 1964-1979 period when Wells G and H were in service the Woburn water pipe network changed very little. In 1970, however, some growth occurred in northeast Woburn and so two separate computer models of the pipe network were needed, one for the 1964-1969 period and one for the 1970-1979 period. Each model is a simplified description of the pipe network. The simplification of the networks was done to reduce the computer time needed for the analysis. Figures 1 and 2 show these simplified pipe networks.

The principal simplification of the pipe networks was the elimination of pipes that were not part of pipe loops. The users' water demands of the omitted "dead end" pipes were assumed to

occur at the nearest pipe junction (node) of the remaining "looped" pipes. A second simplification was the elimination of small pipes whose carrying capacity was less than one-hundredth of that of larger pipes connecting the same pipe junctions. Finally, equivalent lengths of pipe were used to simplify cases where two different diameters were found between junctions and also where a number of small pipes were located in parallel. These simplifications did not appreciably change the results of the computer analysis, and they did significantly reduce the computer time used in finding the water distributions.

The data needed to present the principal water pipes as input for the computer models are given mainly in Table 1. This table gives a detailed list of these pipes' ages, types, dimensions, locations, and interconnections. While this list was well documented(3), the hydraulic roughness of the pipes was not. Therefore data(3) from 1983 pressure tests of fire hydrants, done for the ISO Commercial Risk Services Company, were used to "adjust" the pipe roughness values. This adjustment was a part of the calibration process of the computer models. That process is explained later in section IIb of the report.

The City of Woburn does not have water meters for all its water consumers and did not preserve the 1964-1979 readings of the meters it did use. Residences are charged a fixed annual fee for water, while large commercial and industrial users have meters which are read twice each year. Meter readings in 1984 accounted for 21 percent of the water supplied. In order to estimate the residential water use an average water consumption of 370 gallons

per day per residence was combined with the addresses of the residential consumers. Although there was some evidence(3) of significant leakage flows, because of their uncertain location no attempt was made to include the spatial distribution of leakage in modeling the user demand distribution. Thus the water bills of 1984 were used to prepare a street by street distribution of water consumption. This distribution was then simplified by subdividing Woburn into nearly fifty user demand areas with each corresponding to a representative water supply pipe junction (node) in its area. The boundary between two user areas was placed in the middle of the pipe connecting the two neighboring pipe junctions. Thus each user's residence was roughly assigned to the pipe junction from which its water came. Table 2 gives the street list for each user area and Figure 3 relates the user demand areas to the pipe network and the principal streets of Woburn. The locations of wells, reservoirs, and non-residential pipe junctions are other nodes which are also shown in this figure.

Although Table 2 is the principal description of the user demand areas, the study of adverse reproductive outcomes will locate residences in Woburn through geocodes, a numerical system of geographic location. Such numerical systems also permit computer graphics presentation of the exposure results. Since the following coding of the user demand areas is only approximate, the street lists of Table 2 should be used to determine the demand area of any particular residence.

The author has obtained through the University of Massachusetts Computing Center the numeric codes assigned by the U.S. Census Bureau to describe Woburn. In general these codes are composed of polygons (multi-sided geometric areas) representing the shapes of the states, counties, towns, and Census tracts. These geometric data are presented in the Dual Independent Map Encoding (DIME) format in Tables 3 and 4. This DIME format is commonly used for cartographic data files. Each data line in the tables describes a straight line segment of the boundary between two areas identified by state, county, town, and Census tract. In this system Massachusetts is 25, Middlesex County is 017, and Woburn is 270, and these are assigned here as a part of the numeric codes for each line in the maps of the user demand areas. The usual Census tract codes have been replaced by user demand area codes. (Note that, because of the input format of the graphics program, 100 has been added to the number of the user demand areas in Figure 3 in order to form the geocodes for these areas.) The straight line segments of the boundaries between the user demand areas are defined in the tables by the coordinates of their endpoints. The vertical (North-South) coordinate of each point is its latitude in degrees and ten-thousandths of a degree. The corresponding horizontal (East-West) coordinate is a modified longitude in degrees and ten-thousandths of a degree. The longitude modification is a change of reference site from Greenwich, England to somewhere in Iceland. Figures 4 and 5 are computer graphic representations of these DIME format maps of the 1964-1969 and 1970-1979 user demand areas. The exposure results

presented graphically later in this report are superimposed on these base maps of the user demand areas.

An example of description of the straight line segments of the boundaries between user demand areas is given by the following explanation of the first line in Table 3. The first demand area is identified by the state, county, city, and demand area values as " 25 17270 101." Next along the line, the second user demand area is identified by its state, county, city, and user demand area values as "025 17270 103." The two endpoints of the line segment are identified by the coordinates described above, given in the next position along the line by the two pairs of values, "424535 533884" and "424612 533882." The rest of the line gives two identifiers which were chosen by the author to be zero and a line counter. The first six lines of Table 3, taken together, describe the entire boundary of the first user demand area. That area is shown as #1 in Figure 3 and as area #101 in Figures 4 and 5.

In addition to water pipes and consumers there are also reservoirs and pumps in the Woburn water distribution system. While the pipes are full of water except for the rare occasions when there is a local pipe failure, the storage reservoirs have regular emptying and filling cycles. This cyclic variation of the reservoirs' water elevations is due to the nature of the user demand. The people of Woburn use less water at night since most of them are asleep and they use more water in the morning when they bathe and wash clothes. Industrial demand is also heaviest during the day. An examination of the

water level records for the reservoirs show that the maximum and minimum water demands are roughly 20 percent above and below the daily average demand, and that the maximum, average, and minimum demand periods are roughly of equal duration. These cyclic variations were well documented by the water level records(3) of the reservoirs and are included in the computer models by applying the distribution model to the maximum, average, and minimum demand conditions and then averaging the resulting exposures.

During the 1964-1979 period there were eight well pumps and two significant booster pumps in the Woburn water system. As Figure 1 shows, Wells A2, B, C2, and D are clustered near the Horn Pond Reservoir. Well F is located along the pipe that passes west of Horn Pond. Well E is on Lexington Street just east of Cambridge Road. Wells G and H are off Salem Street near Washington Street, in east Woburn. The Horn Pond wells were controlled by throttle valves so that the pump operators could get the desired flow rates, while these wells' operating pressure was set by the nearby Horn Pond reservoir. Therefore the Horn Pond pumps were characterized simply by their flowrate. The other pumps were defined by their characteristic curves which were given in the previous report(3). All of these pumps were metered, thereby providing the data needed to document their pumping histories.

The pumping history of the 1964-1979 period was developed by examining the daily pumping records for all days when either wells G or H were pumping. The number of days each month that

wells E, G , and H worked in their various combinations were calculated based on the hours pumped during each day of that month. Well E was included since the status of well E caused significant differences in the flow pattern in the water system. The individual pumping histories of the other wells did not affect the resulting distribution patterns due to their proximity to the Horn Pond reservoir. Pumping rates for each month were calculated from the volume of water and hours pumped. The monthly average flow rate of the Horn Pond well group, A2, B, C2, D, and well F was also calculated for each month. Finally, the total monthly water supply and the percentage of that supply that came from wells G and H were determined from the daily pumping records. This pumping history data is given in Table 5.

IIb. Computer Models of the Water Distribution

The analysis of a water distribution system is done by applying the basic principles of the conservation of the mass and energy to the water flowing through the system. The mass conservation principle is applied by assuming that the pipes do not leak along their lengths and that the water users take their water out of the system only at the pipe junctions. Water enters the system at the nodes corresponding to the wells and reservoirs. Thus the net flow rate into each user demand node must equal the user demand, Q_d, of that pipe junction. In most analyses the user demands are known and the flow rates, Q_i, in the pipes connected to each demand node are the unknowns to be

determined by the analysis. Mass conservation for each demand area is given by an equation of the form,

$$Q_1 + Q_2 + Q_3 = Q_d \quad . \qquad (1)$$

The energy conservation principal is expressed in terms of height, usually referred to as "head." Potential energy is described by the elevation, Z, of the centerline of the pipe above mean sea level. The kinetic energy per unit weight is given by the square of the flow velocity divided by twice the acceleration of gravity, $V^2/2g$. The pressure energy per unit weight is given by P/gamma, where P is the pressure and gamma is the specific weight of the water. All these three kinds of energy are combined to form H, the "total head" or mechanical energy per unit weight by the equation,

$$H = Z + V^2/2g + P/\text{gamma} \quad . \qquad (2)$$

The energy principal is applied to all the pipes of the network by requiring that the energy lost to friction, h_L, is the same as the energy differences between the junctions at the upstream and downstream ends of each pipe, as shown by equations of the form,

$$h_L = H_1 - H_2 \quad . \qquad (3)$$

This energy lost to friction can be calculated with one of the various empirical formulas that relate the loss to the flow rate through the pipe, the length and diameter of the pipe, and the roughness of the inner wall of the pipe. The formula used here is the Hazen-Williams equation,

$$h_L = 4.73 \; L \; Q^n \; / (\; C^n \; D^m) \quad , \qquad (4)$$

where L is the length of the pipe, D is the diameter of the pipe, n is 1.852, and m is 4.87.

A corresponding energy analysis describes how the well pumps and reservoirs supply the total head that drives the water into the pipe network.

The result is a large number of linear and non-linear equations which must be organized and solved to determine the flow rates through each pipe and the total head at each pipe junction. Table 6 summarizes the variables needed for this analysis and gives typical values and error levels for the data used in this study. The two mathematical methods commonly used to do this are the successive linear approximation method and the Newton-Raphson method. More detail on the theory of water distribution system analysis can be found in elementary fluid mechanics textbooks and in Analysis of Flow in Pipe Networks by Roland W. Jeppson (4).

The computer program used for the analysis of the Woburn water distribution system is called NETWK. It was developed by Professor Roland W. Jeppson of Utah State University in association with the consulting engineering firm, CH2M-Hill of Corvallis, Oregon. The program is used by the author under a license from that firm.

The computer modeling of the water system began with a 1983-4 calibration process, comparing the 1984 model with total head values based on a set of 1983 ISO data and other data for the water system on the days of those ISO field tests(3). The ISO tests were done during two types of water demand, the normal demand on the test dates and the demands produced when individual fire hydrants are opened and their outflows are added to the

normal demand. The total head values based on the ISO data for normal demand are shown in Figure 6. They indicate that the total head in Woburn was roughly 250 feet with higher values near the water sources and lower values near the water users. Southwest Woburn has even higher values due to the Cambridge Road booster pump.

The initial model had a total user demand of 3,411 gallons pe minute and the same roughness coefficient, C = 80, for every pipe. This initial model was used to calculate the flow pattern and the total head distribution, shown in Figure 7, of the water system. First, a comparison between the supply and demand flows of this computer model and the 1983 test's flow data was made. The model's 1984 user demand estimate totalled only 3,411 gpm while the 1983 supply from the city's wells and the MDC, combined with reservoir outflow, gave a total supply of 5,450 gpm. The 1984 user demands were checked and corrected, but the demand total was still considerably less than the supply. This small demand indicated that an "adjustment" should be made through the use of a scaling factor, 1.4, for the total water demand.

A second adjustment was made by comparing the reservoir flow The model's results indicated an excessive transfer from the Horn Pond reservoir to the Rag Rock tank. The values of the pipe roughness, C, were changed to correspond to roughness values based on the age of the pipes, with older pipes having C as low as 40 and newer pipes as high as 120. The increased roughness along Main Street eliminated the transfer but dropped the total head in northwest Woburn well below the ISO values. Therefore,

the C value on north Main Street was raised to 80, and a computer model of the reservoir flows and total head distribution was obtained that was comparable with the calibration data.

Finally, since the calibration data included measurements with individual hydrants open, the model was compared with key hydrant-open data. Based on these comparisons, the C values of pipes between these hydrants and their water sources were changed to reduce the difference between data and model. The total head values of the final computer model are shown in Figure 8.

A measure of the error level of this ISO calibration process is obtained by comparing the total head distribution of the ISO measurements with that of the computer model after all the "adjustments" to user demand and pipe roughness were made. The average head difference of 28 data points and model values for total head was 0.9 feet, while the root-mean-square head difference was 6.2 feet.

The computer model of the 1983-1984 water system was field tested in 1985 to determine its validity and accuracy(3). Two different model predictions were checked, 1) the pressure distribution near Salem and Washington Streets when a hydrant near that intersection was flowing and, 2) the pressure and fluoride distributions throughout Woburn when no fire hydrants were flowing. An assumption of the computer predictions was that the operation of the water system on the day of the field test would be typical of normal operation, but such was not the case. Therefore a re-prediction was made to correspond to that day's unusual operating conditions. Good agreement was found between

the 16 field measurements and the computer model re-prediction. The error levels of the total heads have a mean value of 0.1 foot and a root-mean-square value of 7.5 feet. More details of the pressure validation analysis is available in the previous report(3). The fluoride distribution prediction and measurements are more descriptive of the distribution of water from Wells G and H. This fluoride validation is discussed further in the next section of this report.

The 1983-1985 water distribution model was next modified to produce models of the water system during the two periods, 1964-1969 and 1970-1979, when the contaminated wells were in service. User demand areas and pipes were deleted or changed to reflect the history of the development of the city. These changes had a large effect on the northeast section of Woburn but not on the overall water supply system. The 1970-1979 model was compared to ISO hydrant data from the years 1970, 1972, 1974, and 1975 to check the validity of the modified model(3). Similarly, the 1964-1969 model was compared to ISO data from 1965 and 1969. The mean pressure differences shown by these comparisons were small, 3.6 feet for 1970-1979 and -3.1 feet for 1964-1969, as was the root-mean-square value of 9.5 feet for 1970-1979, but the root-mean-square value of 16.3 feet for 1964-1969 was high. Since the operating conditions during these tests were unknown, corrective action lacked direction and so no changes were made.

The report(3), "Water Distribution in Woburn, Massachusetts," presented the application of the computer models reviewed in this section to produce thirty-six exemplary flow patterns.

Eighteen patterns showed water distributions with well E on and eighteen with E off. Each group was made up of flows for maximum, average, and minimum percentages of flow from wells G and H, for maximum, average, and minimum values of daily user demand, and for both the 1964-1969 and 1970-1979 periods. That report did not include the calculation of the mixing which occurred at some pipe junctions but simply divided Woburn into three zones for each flow pattern: Zone A with no G or H water, Zone B with G and H water, and Zone C with some undetermined mixture of G and H water and water from the other wells. In Sections III and IV of this report the determination of the Zone C mixing is considered.

III. Wells G and H Exposure Model and its Validity.

The computer program for the mixing of the water from wells G and H was first developed and used by the author as a part of the effort for a report(5), "The Great Oaks Water Distribution Study," prepared for the California Department of Health Services. This study of the Great Oaks Water Company's pipe network, wells and user demand areas followed the method of the previous Woburn study(3) by applying the NETWK computer program to analyze the flow patterns in that water district. The output of the NETWK water distribution program determined the flow rate in each pipe of the water system. Then the author's computer program for the mixing process was applied to these flow patterns.

The analysis of the contaminant movement and mixing in the water distribution is done by applying the principle of mass conservation to the mass of pollutant in the water system. The mixing process was applied to these flow patterns by assuming that the pipes' flow rates were not changed by the addition of small amounts of dissolved contaminants to the water. The pollutants were assumed to move through the pipes with the water without changing their concentrations, and at each pipe junction the inflows were assumed to mix completely due to the turbulent nature of the pipe flow. The mass conservation equation for this mixing at junctions has the form,

$$P_1 Q_1 + P_2 Q_2 = P_3 (Q_3 + Q_d) , \qquad (5)$$

where P denotes the pollutant concentration, pipes 1 and 2 are inflow pipes to the junction, and pipe 3 is an outflow pipe from the junction. The consumers of water in each user demand area

were assumed to get the mixed water of that node and all the outflow pipes from those pipe junctions were assumed to carry the mixed water to the farther points of the pipe network.

The spread of water from a particular well is determined by setting P = 1.0 for that water source and P = 0.0 for the other sources. Then the computer program sequentially examines the inflows to the nodes downstream from the sources and applies equation 5 to calculate the outflow concentration from those nodes, going from node to node until all the user demand areas are analyzed.

In the present Woburn study the pollutant mass conservation principle and these same assumptions are applied through the same computer program for the mixing process. Before looking at more details of the exposure calculations that apply the NETWK and mixing programs to the 1964-1979 distributions of water from Wells G and H, the affects of the quality of some of the data and the field tests of the two previous studies are now examined to provide some insight into the validity and accuracy of these exposure index calculations.

IIIa. Analysis of Data Quality

The pipe network data in Table 1 describes well all the hydraulic aspects of the Woburn water pipes except their interior wall roughness. This roughness, usually quantified for each pipe by a roughness coefficient, "C", describes the frictional resistance to flow through that pipe in accordance with equation 4. Since directly measured values of C for the pipes

were not available, the computer model's C values were calibrated indirectly from the ISO water pressure data. A sensitivity analysis for these roughness coefficients was suggested by this report's Pear Review Committee (see Appendix A) and so the following analysis was done to show the affects of changing the computer model's C values.

The month of October, 1974 was chosen as the test month since it is representative of the months with the larger areas of exposure to Wells G and H. The pipe roughness coefficients were changed in two ways, randomly and systematically, and the resulting changes in the exposure indices for the user demand areas were calculated. These changes were then examined to determine the affects of the C changes on the exposure indices.

The two sets of random changes in the roughness values were made by adding or subtracting a random integer between -18 and +18 from the roughness values determined by the calibration process. The probability of any of these integers was constant except that zero was twice as likely as any other value since the random number generator used both plus and minus zero. The mean change in both random sets was roughly +2 % of the mean C value, and the standard deviation of the changes was roughly 12 %. These random values are given in Table 7.

The two systematic changes in the roughness values were made by multiplying the calibrated C values by either 1.25 or 0.75. These changes either decrease or increase the frictional resistance of the entire pipe network and make it easier or harder for the well pumps to get the water from the wells to the water users.

The computer model for October, 1974 was run for each of these four sets of pipe roughness changes. The changes in the exposure indices resulting from these four computer calculations are given in Table 7. The average change in the exposure index was less than one percent of the maximum exposure index. Further, three of the four standard deviations of the changes in the exposure index were roughly one percent of the maximum exposure index and the fourth value (3 %) would also have been roughly one percent except for the affect of the exposure index change in one demand area, Node Number 34. That change is underlined in Table 7. This one area's index was more sensitive to variation in the pipe roughness.

In general the pipe roughness changes did not affect the size or locations of the areas which received water only from the Horn Pond wells (index = 0.0) or only from Wells G and H (index = 1.0). Even in the boundary zone where mixing of the waters from the two sources occurred, the pipe roughness changes of ten to twenty five percent caused exposure index changes of only one to three percent. The one sensitive demand area, Node 34 with 3 %, was located in a part of southeast Woburn where the spatial gradient of the exposure index was very high. This sharp gradient can by seen in Figure 20. Overall, this analysis has shown the exposure index to be insensitive by an order of magnitude to changes in the pipe roughness.

The spatial and temporal distribution of the water consumption among the nearly fifty user demand areas is a second set of data whose reliability was questioned by the Peer Review Committee (see Appendix A). In preparing the computer model, the 1984 water bills were the data source for the metered, industrial consumption (21 %) and also the source of data on the locations of the unmetered, residential water users. An approximate monthly average water use of 370 gallons per day was assigned to all residences in each user demand area. Since the exposure index was calculated as a monthly average, the monthly average water supply of the wells was used to approximate the average demand of the daily cycle. Leakage was assumed to be uniformly distributed throughout the city. The daily cycle of more water use during the day and less use late at night was approximated by three steady values: high demand, average demand, and low demand. The validity of the last two approximations is now examined by looking at the one water leakage study available and at three weeks of indirect data for the daily cycle of water demand.

The water leakage survey was done in 1980 by Pipe Line Testing Service, Inc. The survey team placed the transducers of their electronically-amplified, sonic, leak detectors on fire hydrants and water main valves throughout the water distribution system. The sound of the escaping, high-pressure water resulted in the prediction that there were 82 underground leaks. The location of these leaks is

approximate, and so only 38 of the 82 leaks were promptly repaired. The flow rates of the leaks were estimated based on the surveyors' experience. Accordingly, there was " a minimum of 20 gpm saved for main leaks, 10 gpm for service leaks, 3 gpm for hydrant leaks, and 30 and 5 gpm for the two miscellaneous leaks.

8 main leaks at 20 gpm.....	160 gpm
65 service leaks at 10 gpm.....	650 gpm
7 hydrant leaks at 3 gpm.....	21 gpm
1 miscellaneous leak at 5 gpm.....	5 gpm
1 miscellaneous leak at 30 gpm.....	30 gpm
	1238 gpm

This represents a savings of approximately 1,700,000 gallons per day,..." Unfortunately the inaccurate addition and the low repair rate, finding only 38 actual leaks, do not inspire confidence in the leakage estimates of this report. Nonetheless, this was the limited information available and so Table 8 and Figure 9 show the estimated flow rates and locations of these leaks. The report supports only the general conclusions that there was substantial leakage and that it was widely distributed throughout Woburn.

The daily cycle of water demand was examined indirectly by analyzing the flow rates produced by the supply wells and the rate of change of the volume of water in the Woburn storage tanks. Whenever the water elevation in the tanks is

constant, the water demand is then equal to the supply from the wells. During the daytime peak flow, the rate at which water flows out of the storage tanks is added to the well supply to fulfill the demand. Late at night, the rate at which water flows into the tanks is subtracted from the well supply to calculate the minimum demand. The daily cycle of the peaking factor is obtained by dividing the instantaneous water demand by the daily average demand.

In order to simplify this peaking factor analysis, two weeks were chosen when the supply wells were running constantly nearly all week. One summer week with a high average demand and one winter week with a low demand were selected, June 21-27, 1970 and December 15-21, 1974. The week used in the computer model calibration, October 30 - November 5, 1983, was also studied.

While there were four water storage elements in the Woburn water system, the Zion Hill and Whispering Hill storage tanks and the Horn Pond reservoir were maintained at nearly constant water levels by the water level sensors and pump controls. The author's previous report(3) shows two typical water level charts demonstrating this constancy. Thus, the daily demand cycle is seen principally in the water level variation of the Rag Rock water storage tank. The water elevation chart for June 21-27, 1970 is presented in Figure 10, and the charts for the other two weeks of this peaking factor analysis are in the previous report and its appendices(3).

The three weeks of peaking factor analysis was done by looking at water elevation changes during both four-hour and eight-hour intervals. The intervals were chosen to represent the periods with high, average, and low water demands. The low demand period of the early morning was followed by a near average period near dawn and then the daytime high demand period, followed by another near average period in the evening and then a return to the nighttime low. Table 9 shows the results of these calculations, the peaking factors for each individual four-hour or eight-hour interval. Sometimes, the poor data quality prevented the calculation of a peaking factor, and those times are shown in Table 9 with an X. The average values and standard deviations of these three weeks of peaking factors were also calculated and are included in Table 9.

The individual four-hour peaking factor values had considerable variation, from a high of 1.41 on Monday, October 31,1983 to a low of 0.61 on Monday, December 16, 1974. Most days had high peaking factors between 1.1 and 1.3 with low peaking factors between 0.7 and 0.9. However, on Friday, November 4, 1983 the peaking factor was nearly constant all day. This highly scattered data pattern for the four-hour intervals produced the high standard deviation shown in Table 9. The average values for each week are graphed in Figure 11. They show that the peaking factor is typically a little above 1.2 between 9 AM and Noon, a little below 0.8 between Midnight and 5 AM, and nearer to the average value of 1.0 the rest of the time.

The eight-hour interval analysis smoothed out these irregularities and produced results with less variation. The highest eight-hour peaking factor was 1.29 on Sunday, December 15, 1974 and the lowest was 0.71 on Monday, December 16, 1974. The resulting average values support the previous report's use of a high peaking factor of 1.2 and a low of 0.8.

IIIb. Great Oaks Validation Tests

In the Great Oaks study(5) the water distribution and mixing models were checked by using the models to predict the spatial distribution of the fluoride concentration that would result if fluoride with P = 2.00 parts per million were injected at Well 16, shown as GO 16 in Figure 12, and allowed to spread throughout the Great Oaks pipe network. This figure shows the predicted values of the fluoride levels based on the actual test's injection concentration of 1.51 PPM, and Table 10 shows both the original fluoride prediction and the prediction modified by scaling to the actual 1.51 PPM concentration. A field test of the prediction was then made by injecting fluoride at Well 16 and measuring the fluoride distribution that resulted. These fluoride concentrations are given in Figure 13 and also in Table 10.

While both the model and the field test had inaccuracies described in the Great Oaks report, it was clear that the computer models located the boundary between the Well 16 water and the water from the other wells improperly by one node at a few key locations. An error analysis of the predicted and observed

concentrations given at the bottom of Table 10 shows a mean error of 0.03 PPM and a root-mean-square error level of 0.31. The large rms value is due to a few points near the Well 16 water's boundary with large errors. The error bands given in Table 10 show that 64 percent of the predictions were within 0.2 of the measured concentrations.

IIIc. Woburn Validation Study

In the previous Woburn water distribution study(3) a field test of the predicted pattern of fluoride concentrations was only compared qualitatively with the computer model's prediction. At that time mixing analysis was not included in the fluoride calculations. During this study the fluoride concentration prediction is re-examined by doing that mixing analysis. The 1985 fluoride source was the MDC connection on Montvale Avenue in east Woburn. Its fluoride concentration was 1.07 PPM while the other sources of water, Woburn's wells, had only 0.07 PPM.

The qualitative comparison of the prediction, Figure 14, and the field test results, Figure 15, shows that both divide Woburn into three zones, an east Woburn zone with MDC water (1.07 PPM), a southwest Woburn zone with water from the Woburn wells (0.07 PPM), and a northwest zone with a mixture of water from both sources. The prediction indicates that the boundary between the first two zones is located one node east of Main Street except at Mishawum Road and Eaton Street. The field test indicated that the boundary was along Main Street. A similar one node difference in location occurs between the MDC water and the mixed water in north Woburn.

The predicted and observed(two labs) values of the fluoride concentrations are also compared in Table 11. This time the mean error was 0.07 PPM and the rms error was 0.29 PPM. The error bands in Table 11 show that 72 percent of the predictions were within 0.2 of the observed values. These values and the other statistical analyisis shown in Table 11 indicate an accuracy level nearly equal to that given by the Great Oaks validation.

Taken together the two field tests using the fluoride tracer demonstrate that the water distribution and mixing models can predict the location of the boundaries of the area receiving water from Wells G and H within one node. The models can predict mixture concentrations with an average error within ten percent of the maximum concentration and a root-mean-square error within thirty percent of that level. Large errors(100 %) in the mixture concentrations occur at a few points near the fluoride boundary due to one node errors in locating the pipe junctions where mixing occurs, but nearly seventy percent of the predictions are within twenty percent of the measurements.

IIId. Well G and H Exposure Study

In the present study of the exposure to water from Wells G and H the existing water distribution models for the periods 1964-1969 and 1970-1979 are applied to obtain typical water distribution patterns for each month when the wells were pumping. The mixing program is applied to each pattern to calculate the fraction of the water supplied to each user demand area which came from the contaminated wells.

As was shown in the previous Woburn report(3) the flow patterns are not affected by different combinations of wells near Horn Pond, but different flow patterns are produced when Well E is on and when it is off. A typical month has periods with Well E on or off and also has three peaking factors(1.2, 1.0, 0.8) accounting for the maximum, average, and minimum water demands of the daily water-use cycle. The monthly analysis must therefore account for each of these six conditions and requires six water distribution calculations and six applications of the mixing program. A small computer program was developed during this study to combine the six resulting mixtures for each month in proportion to their duration.

The result is an exposure index assigned to each user demand area during the months of the two exposure periods, 1964-1969 and 1970-1979, when the G and H wells were in operation. That exposure index is the product of the fraction of the time during the month when any contaminated water from Wells G and H reached the user area and the fraction of the water supplied to that user area which came from the contaminated wells. The units of this exposure index are months, with a maximum value of one month occurring if Well G is on all month and if the user demand area was supplied with Well G's water during the entire month. Since this method of calculation of Well G and H exposure is the same as that applied to the fluoride mixing prediction, the resulting exposure indices should have the level of accuracy indicated by the fluoride field tests.

During some months, for example August and September of 1967 the six distributions needed for different peaking factors and Well E status were not enough. In these months when Wells G and were operated in different combinations, separate calculations were done for each combination and then either twelve or eighteen different results were combined to produce the monthly average exposure indices of those months.

Table 12 shows the monthly averaging process for May, 1979 as an example of the combination process for the six distribution case. The pumping conditions given in Table 5 and at the top of Table 12 show the fractions of this month that Wells E, G, and H were operating. Wells G and H were operating during 70 % of the month. Well E was on all month. The six columns on the right in Table 12 give the fraction of the water that came from contaminated wells during the parts of the month when the average, maximum, and minimum water demands occurred with Well E off or on. Since Well E was on all month, the monthly exposure is the average of the three exposures for the different peaking factors with Well E on, multiplied by 0.70. The column labeled EXPINDX presents the resulting monthly average exposure index for each user demand area for the month. The maximum index, 0.70 months, is found in the areas of east Woburn. An example of a smaller average exposure index, that for node 32, is given by the calculation,

$$(0.70)\ (0.124 + 0.067 + 0.180) / 3 \quad = 0.087 \quad . \qquad (6)$$

IV. Exposure and Cumulative Exposure Results.

The water distribution and mixing models were used to calculate the exposure indices for all the months when Wells G and H were pumping. The spatial patterns of the exposure distributions are difficult to understand from a list of the exposure values, and so Figures 16, 17, and 18 are presented to show the patterns typical of medium, broad, and small areas of exposure to water from Wells G and H during the 1964-1969 period. The exposure index values for 1964-1969 are given in Table 13. The initial results indicated that street lists of the previous report(3) for user demand areas 33 and 34 were incorrect and so these areas were changed to reflect the initial flow pattern.

In Figure 16 the geocode description of the user demand areas is the base upon which the exposure index results are presented. Different densities of cross-hatching show six ranges of values of this exposure and the areas without any cross-hatching give a seventh. The densities increase with increasing exposure. The five main exposure ranges are roughly equal in size, 0.20 months, and they give the high exposure pattern. In addition two smaller ranges present an order of magnitude estimate of a trace range (0.010 to 0.001 months) and a near zero range (no cross-hatching). The exposures shown are those of June, 1966, a month whose exposure pattern is typical of the median of the 1964-1969 period. The areas of east Woburn along Washington Street and just to the west of that street received G and H water nearly all that month. The areas of southwest Woburn received no G and H water and a band of user demand areas near Main Street received various mixtures of

the G and H water and water from the Horn Pond wells.

A broad area of exposure is shown by the exposure pattern of October, 1967 (Figure 17). It is typical of the largest spread of the water from Wells G and H. During this month the mixture areas near Main Street received a higher fraction of their water from Wells G and H, and trace amounts of G and H water even reached the extreme southwest corner of Woburn. Only a small zone around Horn Pond was unaffected by the water from those two wells.

A small area of exposure is given by the exposure pattern of June, 1969. Thus Figure 18 is typical of the minimum spread of G and H water during the 1964-1969 period. During that June both the high exposure zone and the mixture zone were limited to the demand areas east of Main Street. Water from Wells G and H did not reach northwest Woburn during this month.

The spatial patterns of the exposure distributions of the 1970-1979 period are illustrated by Figures 19, 20, and 21. These figures show patterns typical of medium, broad, and small areas of exposure to water from Wells G and H during that period. The exposure index values for 1970-1979 are given in Table 14. The water distribution model changed in 1970 due to the development of new housing, commerce, and industry, largely in northeast Woburn. This change increased the water demand in that part of the city and so decreased the spread of the G and H water to the rest of Woburn. This effect caused the main difference between the results of the periods 1964-1969 and 1970-1979.

The exposure pattern of March, 1979 is shown in Figure 19. Its medium exposure area is typical of the 1970-1979 period. The areas of east Woburn along Washington Street and just to the west of that street received G and H water nearly all that month. As in Figure 16, areas of southwest Woburn received no G and H water and a band of demand areas near Main Street received various mixtures of the G and H water and water from the Horn Pond wells. This pattern is quite similar to that of Figure 16.

Figure 20 presents the broad exposure pattern of the month of October, 1974. It is typical of the maximum spread of the water from Wells G and H during 1970-1979. While it is similar to the maximum pattern for 1964-1969, shown by Figure 17, in Figure 20 the trace amounts of G and H water do not reach into southwest Woburn.

The smallest zone of exposure for both the 1964-1969 and 1970-1979 periods is shown in Figure 21. That figure gives the exposure pattern for October, 1976. It shows peak exposures only in those areas along Washington Street that are near Salem and Montvale Avenues. During this month the mixing zone reaches both the northern and southern ends of Washington Street, and the zone free of exposure extends well to the east of Main Street.

The cumulative exposure for every month when Wells G or H were pumping was also calculated. This was done by adding the exposure index for each month to the cumulative exposure of the previous month whenever Wells G or H were in operation. The resulting sum is the number of months of exposure prior to the end of the month whose cumulative exposure is being calculated.

The month of October, 1964 was special since as the first month it had no antecedent. In its case the cumulative exposure and the exposure index are equal. These results are presented in Tables 15 and 16. The cumulative exposure for the many months when neither Well G nor Well H was pumping is not listed in these tables since the cumulative exposure for any of those months is the same as that of the previous month in the tables.

Since the goal of this study is to examine the rate of adverse reproductive outcomes as a function of exposure to water from Wells G and H, some examples of the use of Tables 13, 14, 15, and 16 are included here. Assuming that a nine month pregnancy began November 1, 1964 and ended July 31,1965 and that the mother lived in user demand area(node) 66, Table 13 gives exposures of 0.47 and 0.50 months for the first two months of the pregnancy and zero (G and H both off) for the next seven months. Table 15 gives a cumulative exposure for the mother of 1.51 months, but also indicates (by subtraction) a cumulative exposure during the pregnancy of 0.97. If a five month pregnancy of a mother living in node area 35 began March 1, 1976 and ended July 31, 1976 then the exposure values (Table 14) were 0.42, 0, 0, 0.61, and 0.97 months, while the cumulative exposure is 2.00 months. Finally, if a mother lived in user demand area 58 all her life and had children after 1981, then her cumulative exposure (Table 16) before her pregnancies was 64.08 months.

V. Conclusion.

The purpose of this study of the exposure to the water from Wells G and H in Woburn, Massachusetts is to describe when and where that water was delivered to the residences of Woburn. This result will then be compared with occurrences of selected adverse reproductive outcomes through the Health Outcomes Study.

Tables 13 and 14 report the principal results of this study. The water pipe network of the 1964-1969 period of operation of Wells G and H was used to divide the city into 52 hydraulically distinct neighborhoods, and the pipes of the 1970-1979 period were used to give 54 such neighborhoods. Since the wells pumped during 44 months of the 1960's period, Table 13 is a matrix of numbers between zero and one that indicate the fraction of the water, supplied to each user area, which came from Wells G and H during each of the 44 months. Table 14 presents this exposure index for the 54 user areas during the 70 months of the 1970-1979 period. These matrices demonstrate that areas 11, 17, 18, 19, 20, 21, 22, 30, and 31 did not receive a significant amount(0.01) during any month. At the other end of the exposure range, areas 39, 42, and 44 received all their water from Wells G and H whenever either of those wells was operating.

The accuracy of these exposure results was estimated by examining both the fluoride field test results and the ISO fire hydrant comparisons of total head data and computer model calculations. The fluoride tests show that the computer models for present-day water systems can only locate the fluoride boundary within a spatial error of one node, and so predicting

the mixture concentrations in the user areas along that narrow boundary is a difficult task. Tables 10 and 11 indicate that the fluoride predictions had an root-mean-square error level of 30 percent due mainly to a few large errors near the boundary between the zones supplied by the two sources. This indicates that the exposure index values do not have the two significant digits reported in Tables 13 and 14, and that the trace levels shown in Figures 16-21 are only roughly valid. Further, the ISO comparisons in Section IIb demonstrate that the lack of historical data increases the differences between the computer model results and the actual field measurements.

However, even with this high error level, the low mean error and the error band analysis indicate that on average there is a connection between the models' predictions and the field observations. It is the author's opinion that a good estimate of the error level of the models is twenty to thirty percent. Thus the mixture region should only be divided into three sub-regions: 0.0-0.33, 0.34-0.67, 0.68-1.00.

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Figure 1 Woburn, MA 1964–1969 Pipe Network with pipe numbers and diameters

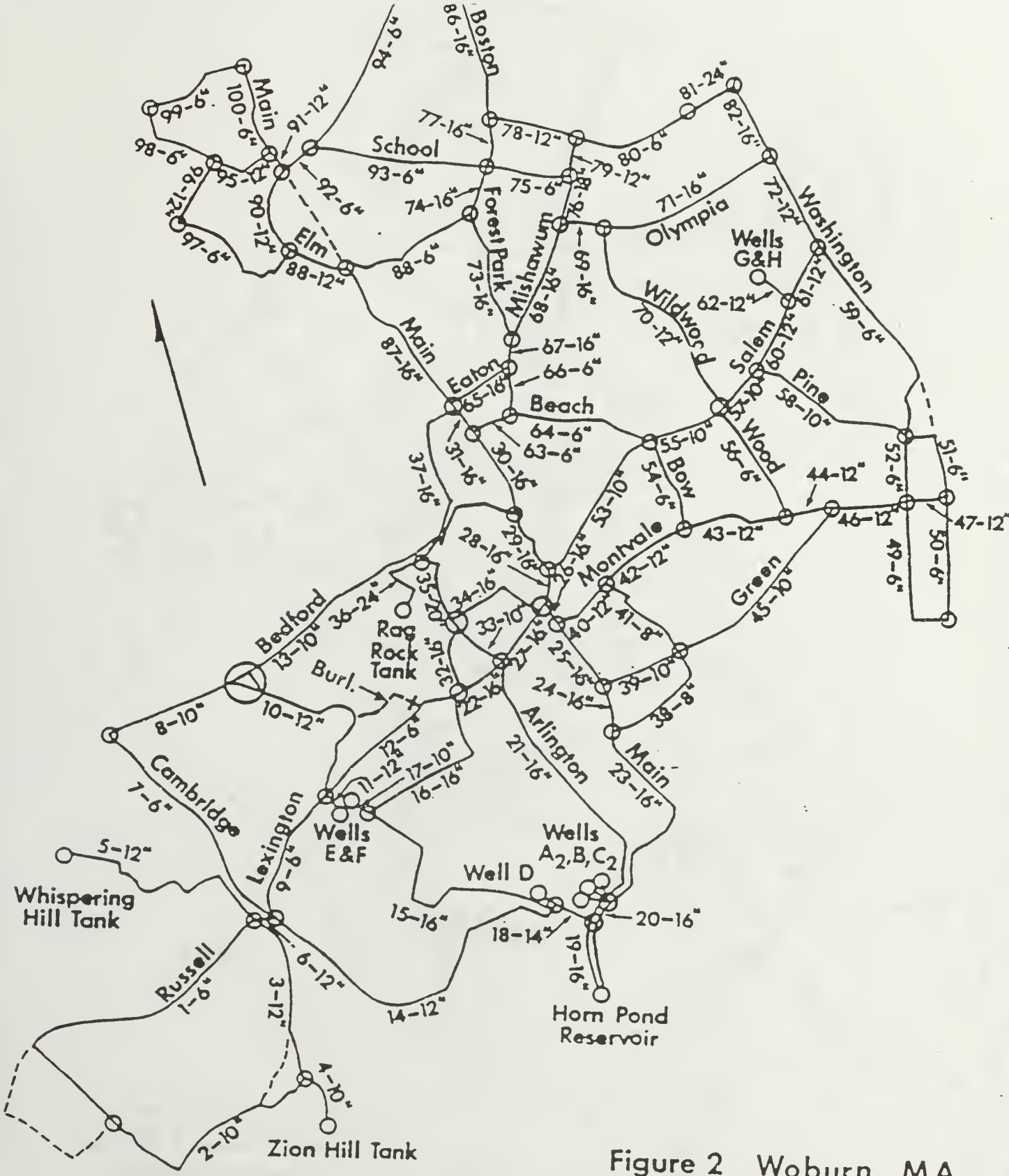

Figure 2 Woburn, MA 1970-1979 Pipe Network with pipe numbers and diameters

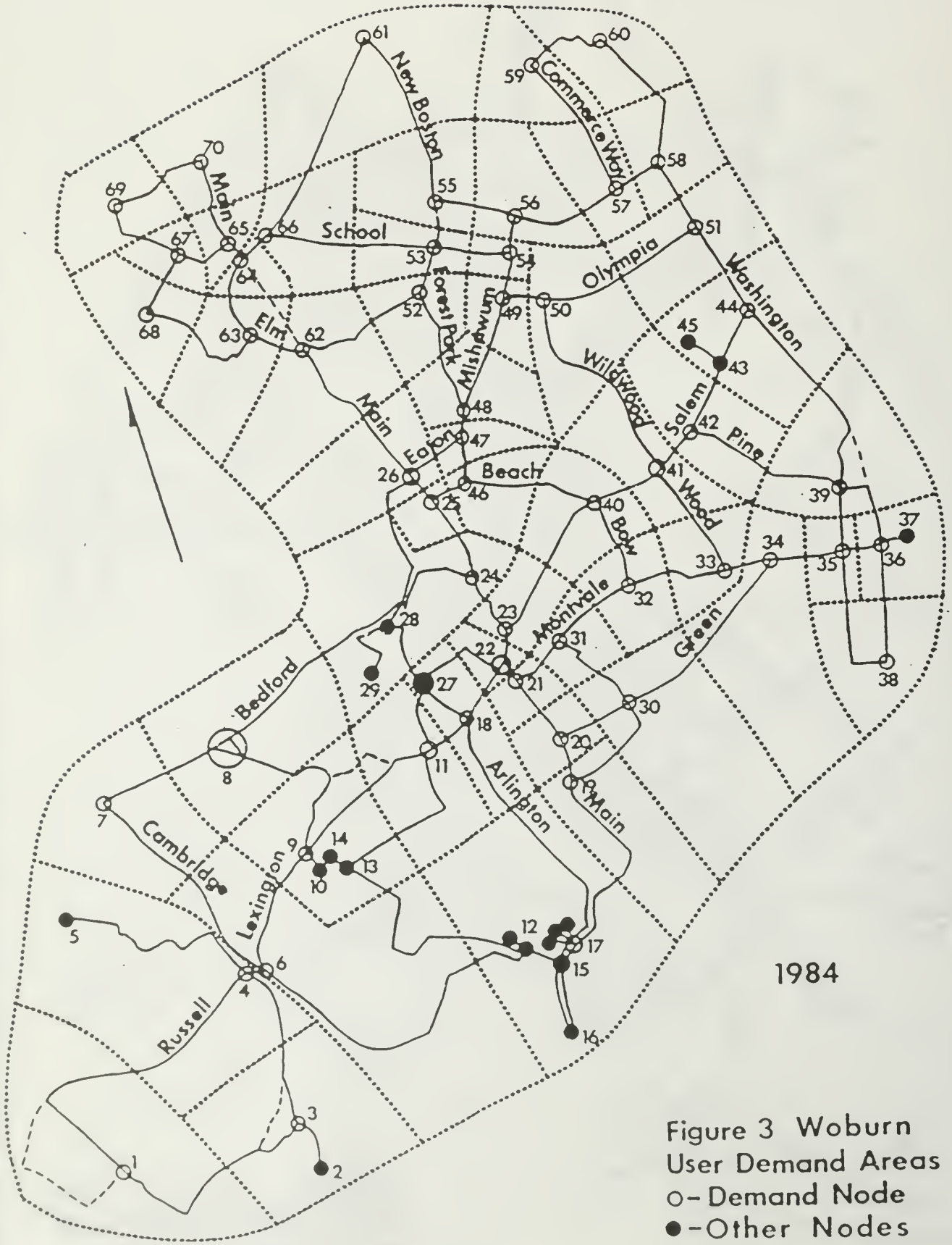

Figure 3 Woburn
User Demand Areas
o- Demand Node
●-Other Nodes

FIGURE 4. 1964–69 WOBURN USER DEMAND AREAS

FIGURE 5. 1970–79 WOBURN USER DEMAND AREAS

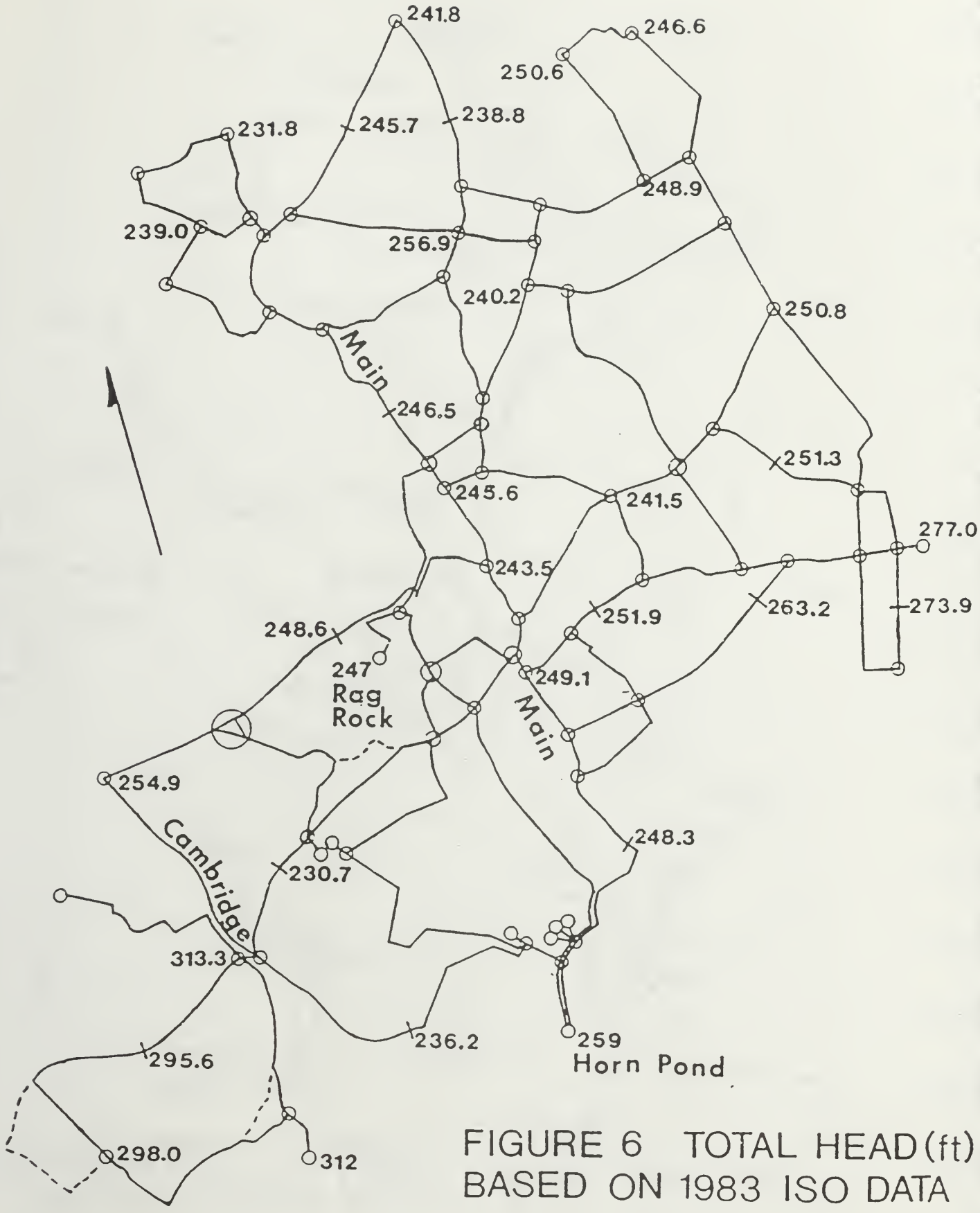

FIGURE 6 TOTAL HEAD (ft)
BASED ON 1983 ISO DATA

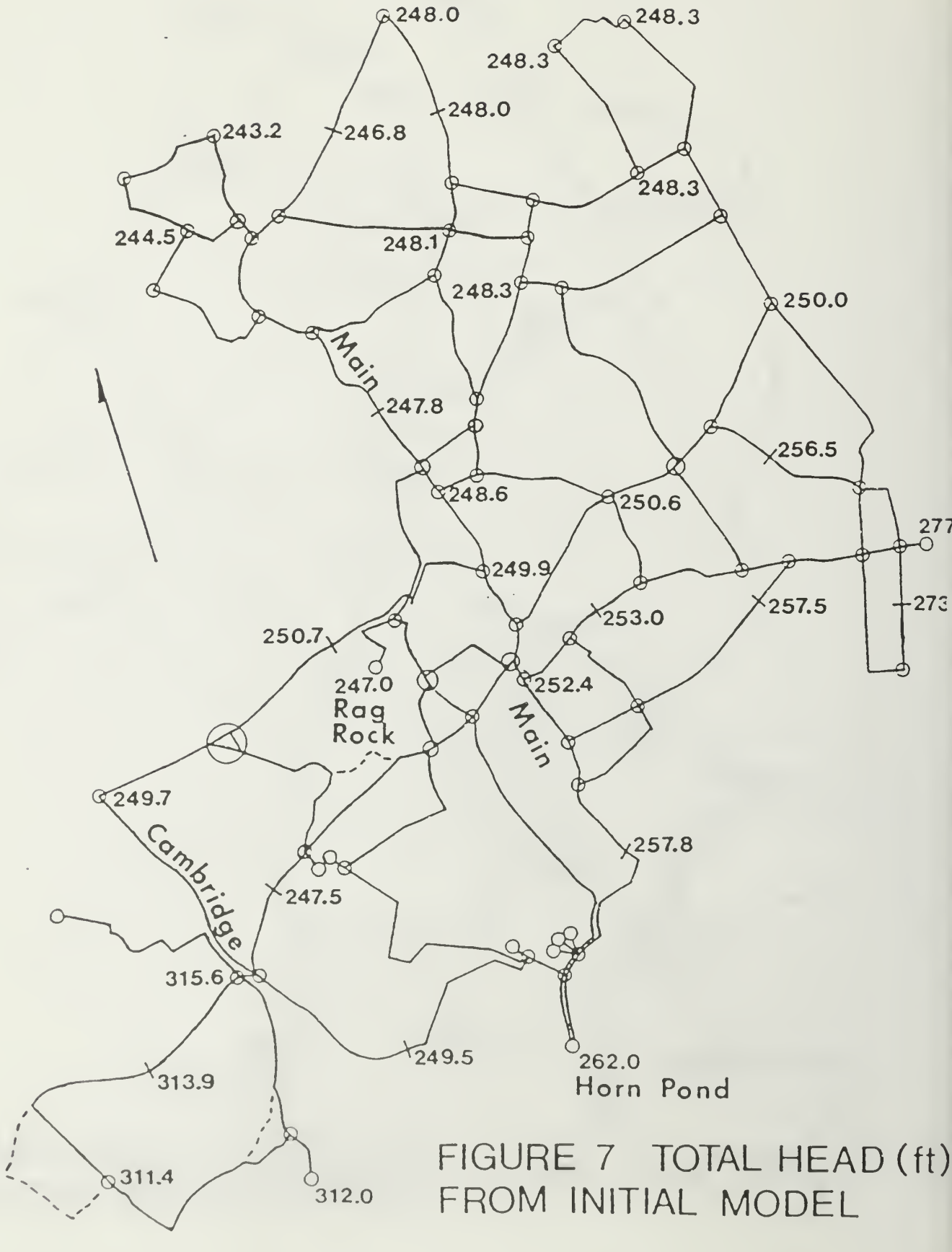

FIGURE 7 TOTAL HEAD (ft) FROM INITIAL MODEL

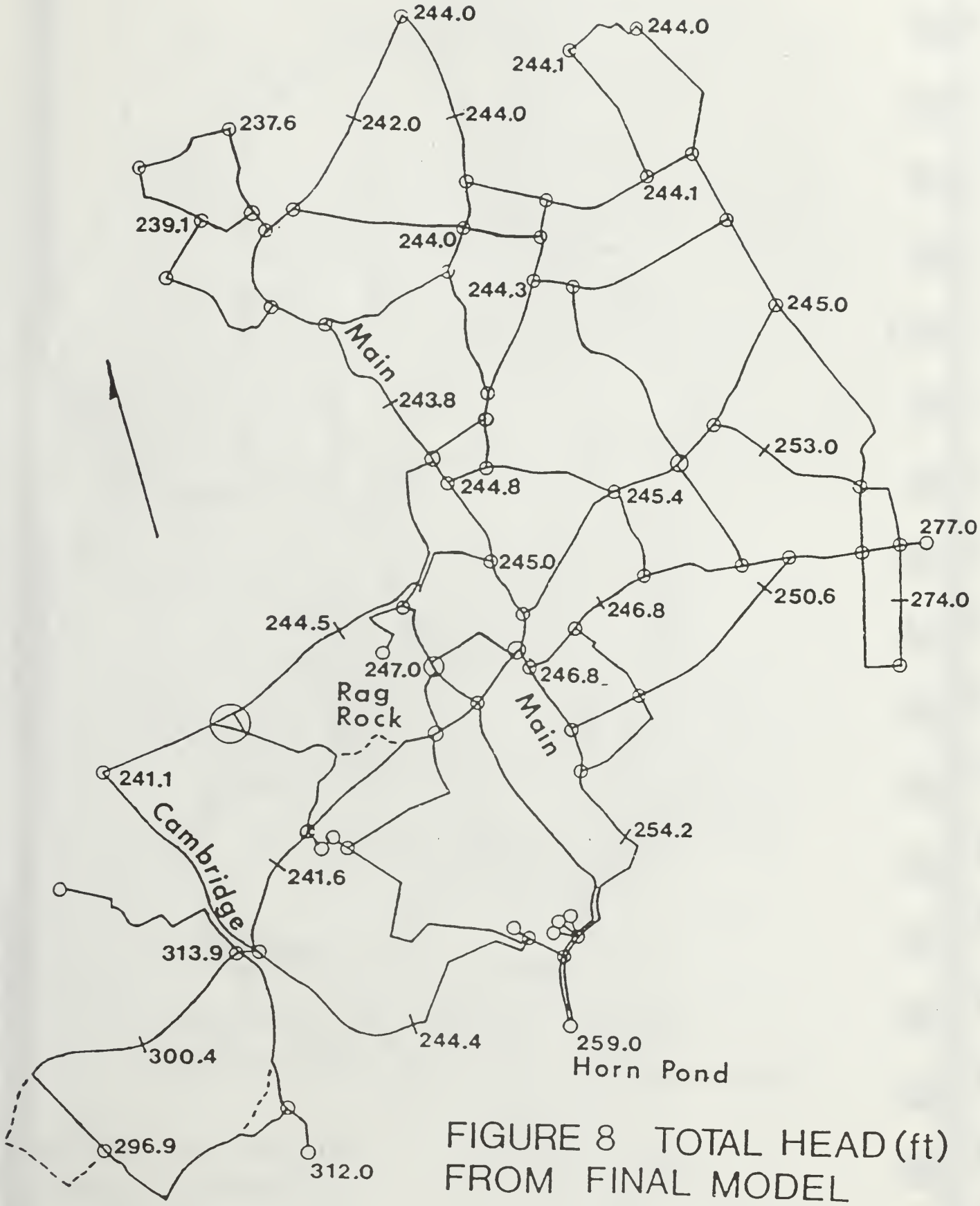

FIGURE 8 TOTAL HEAD (ft) FROM FINAL MODEL

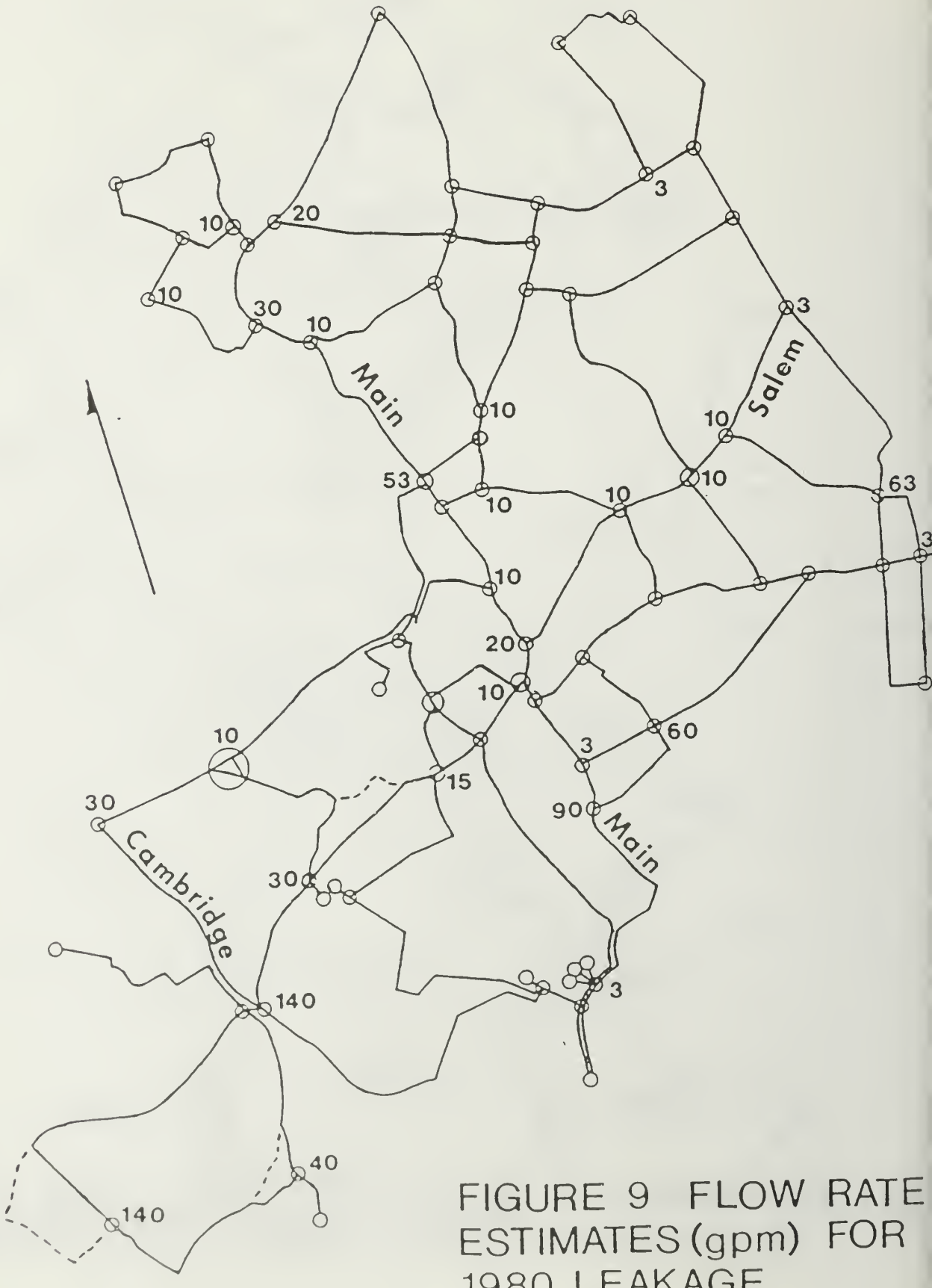

FIGURE 9 FLOW RATE ESTIMATES (gpm) FOR 1980 LEAKAGE

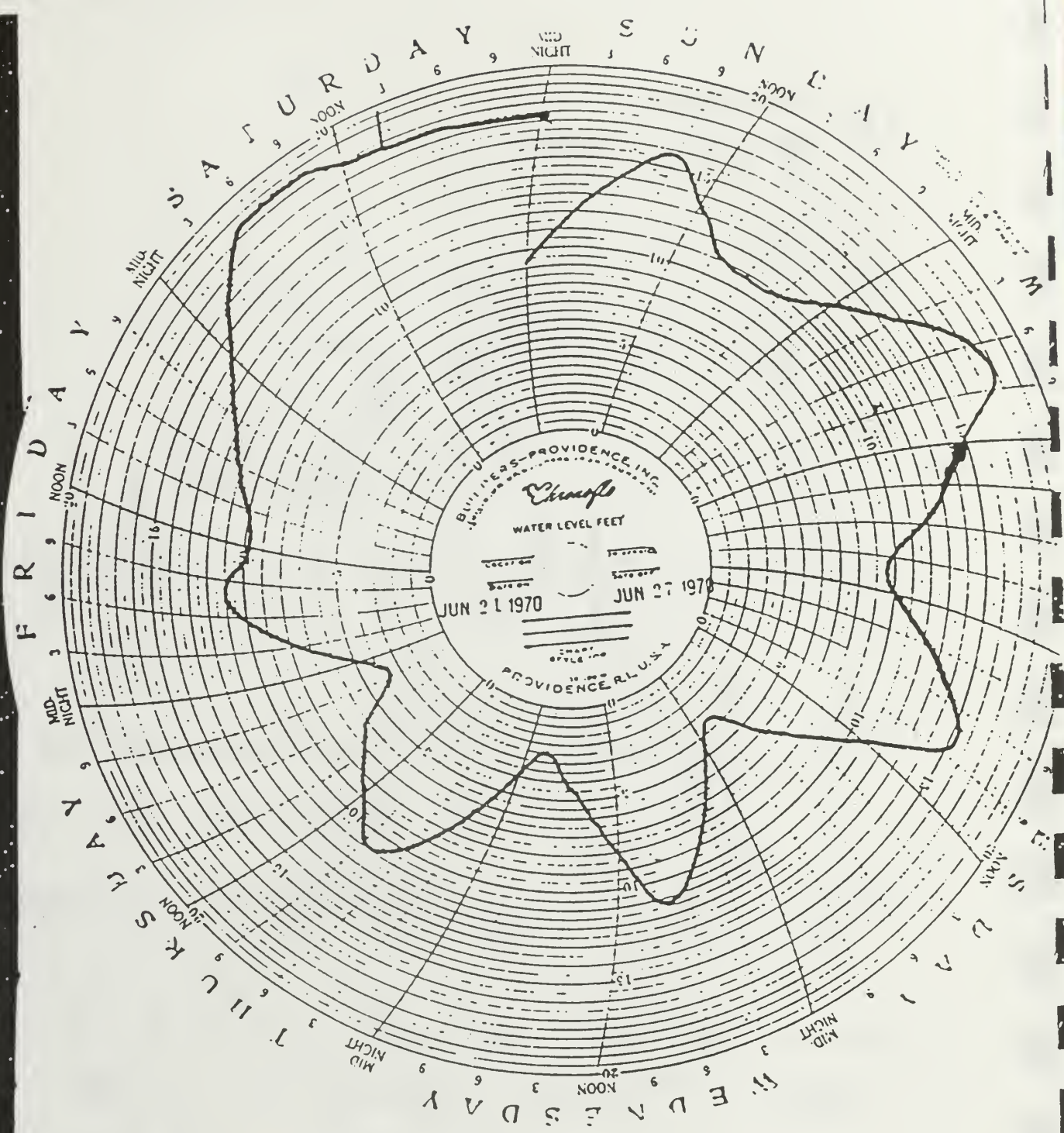

FIGURE 10 WATER LEVEL VARIATION IN RAG ROCK TANK

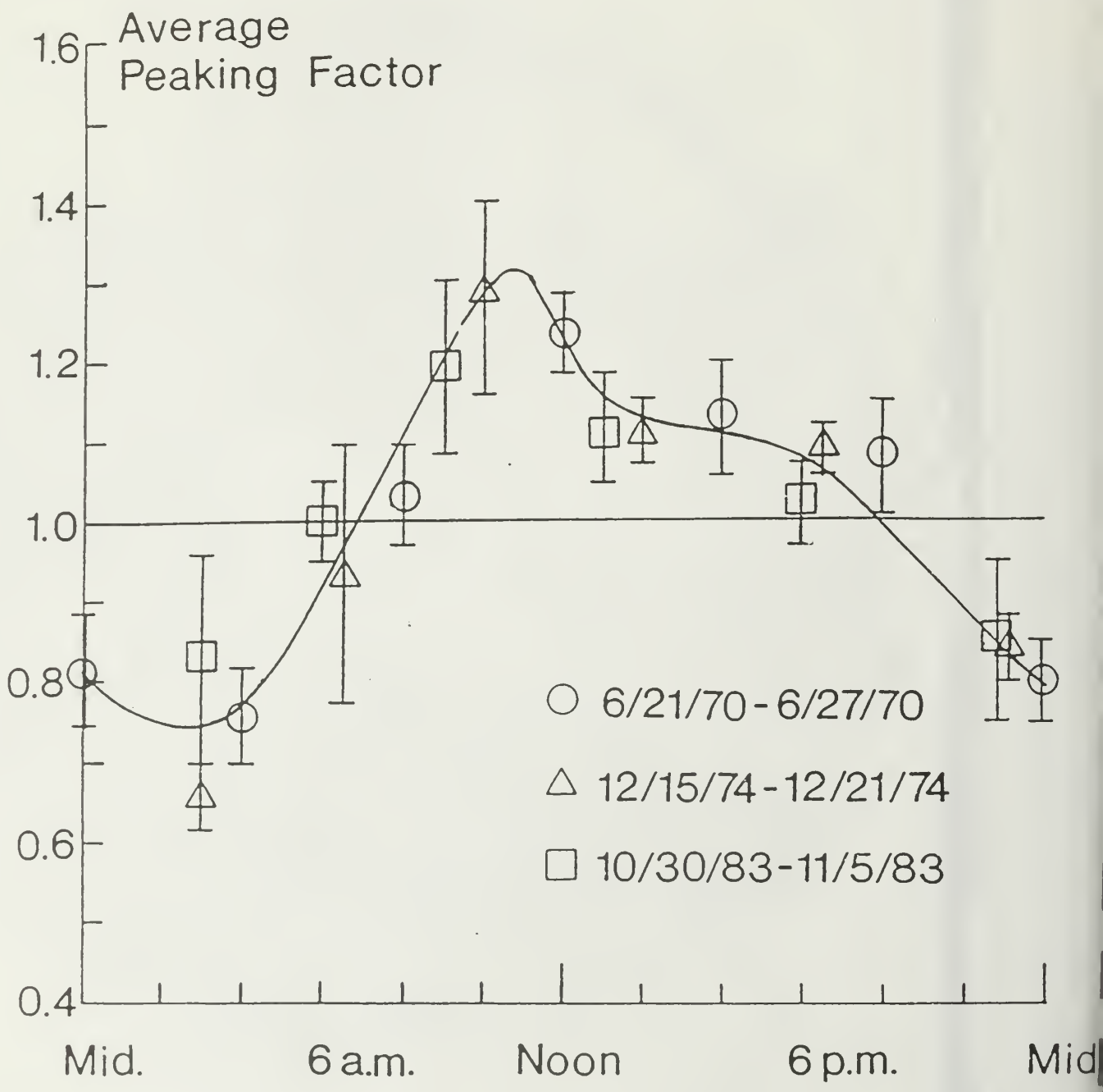

FIGURE 11 DAILY CYCLE OF PEAKING FACTOR

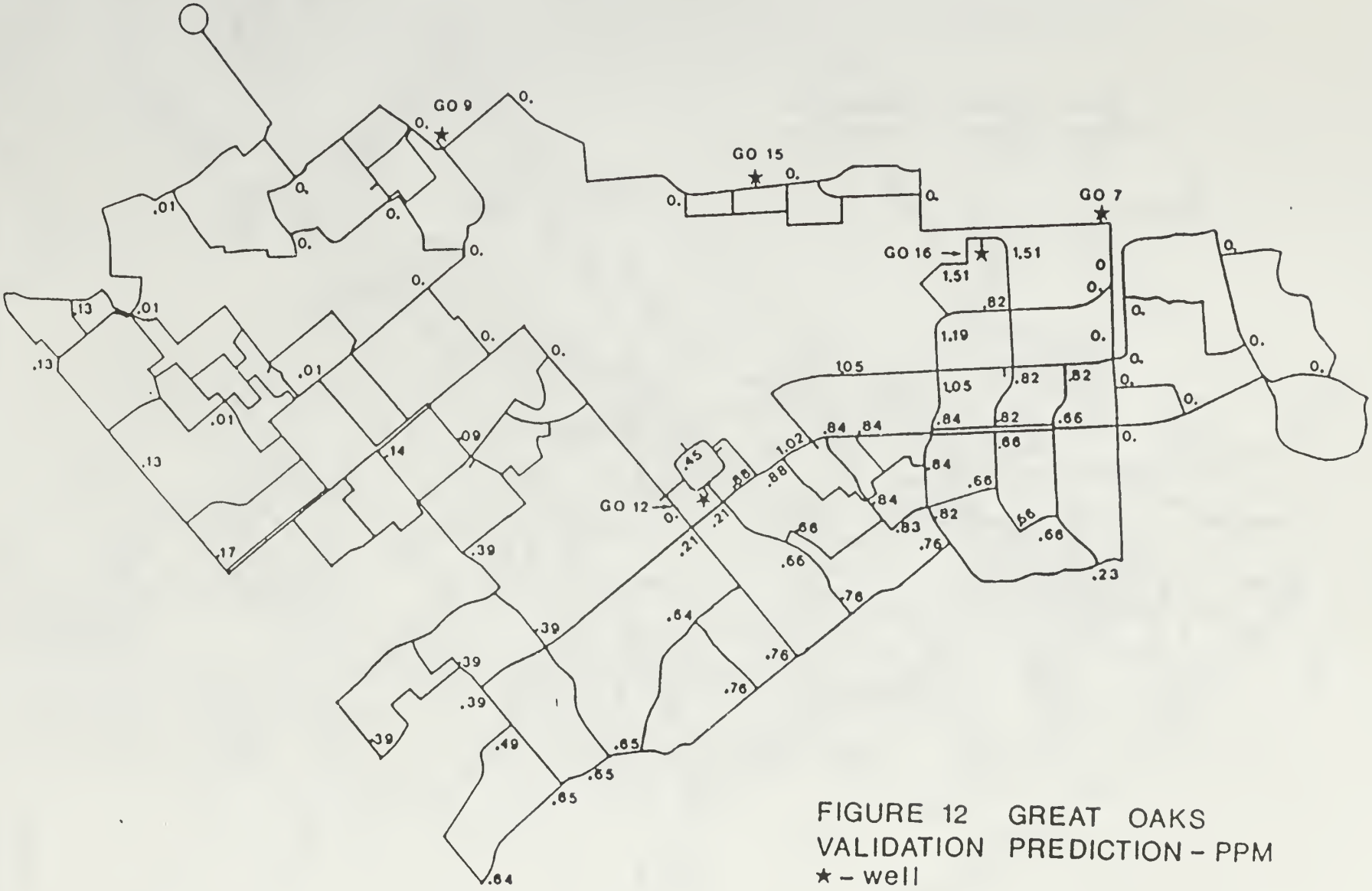

FIGURE 12 GREAT OAKS
VALIDATION PREDICTION - PPM
★ - well

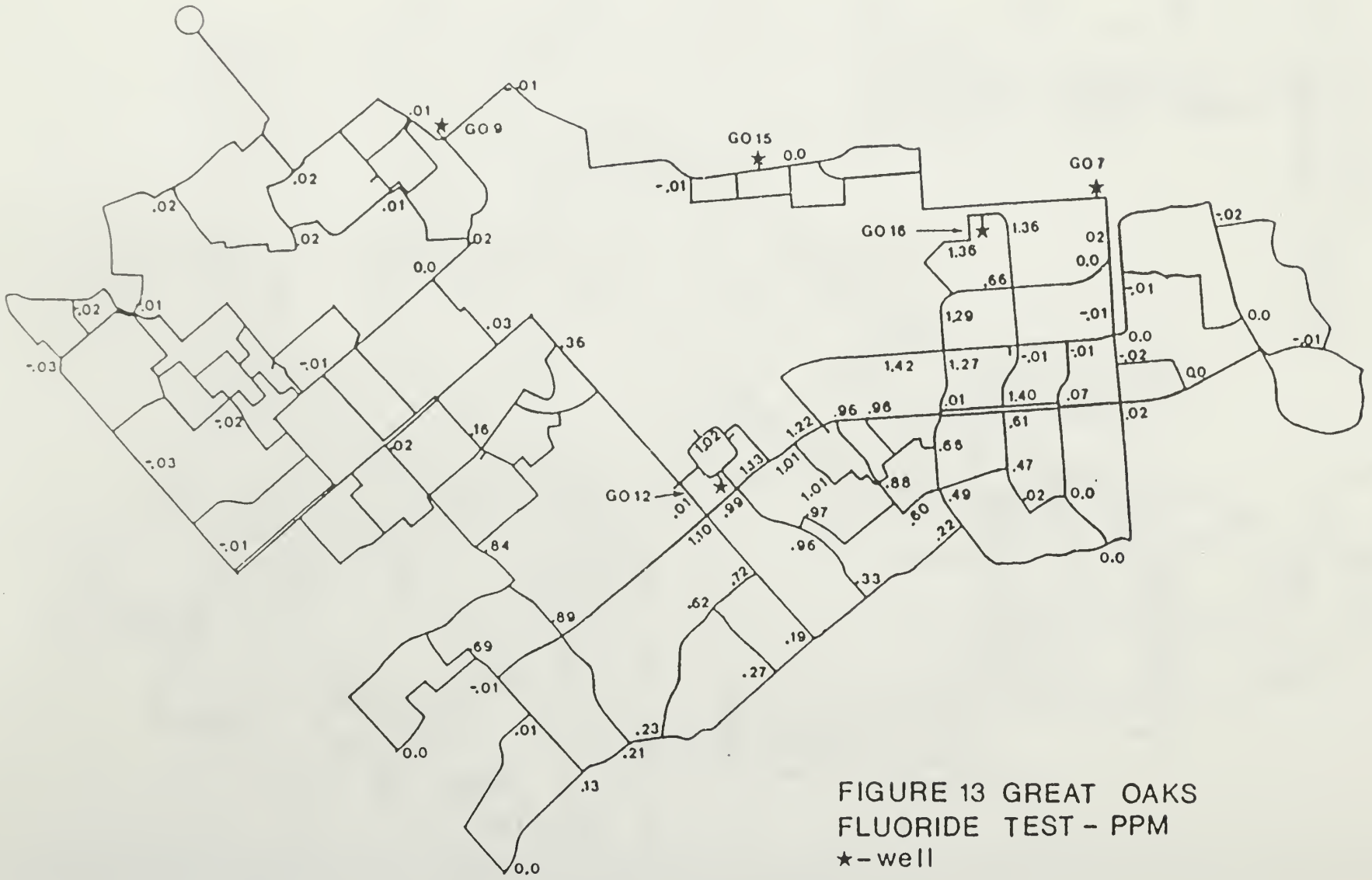

FIGURE 13 GREAT OAKS
FLUORIDE TEST - PPM
★ - well

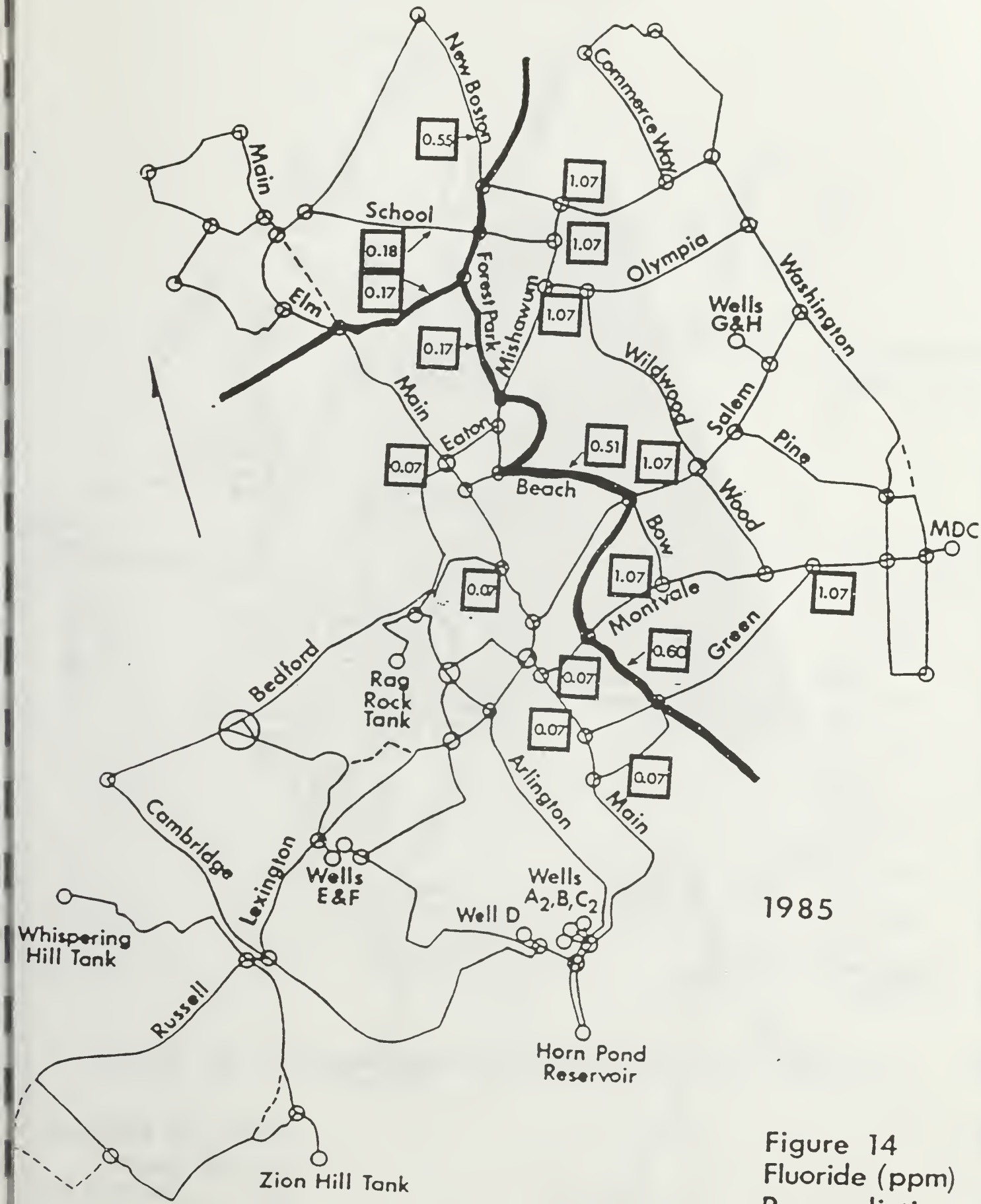

Figure 14
Fluoride (ppm)
Re-prediction
for Woburn

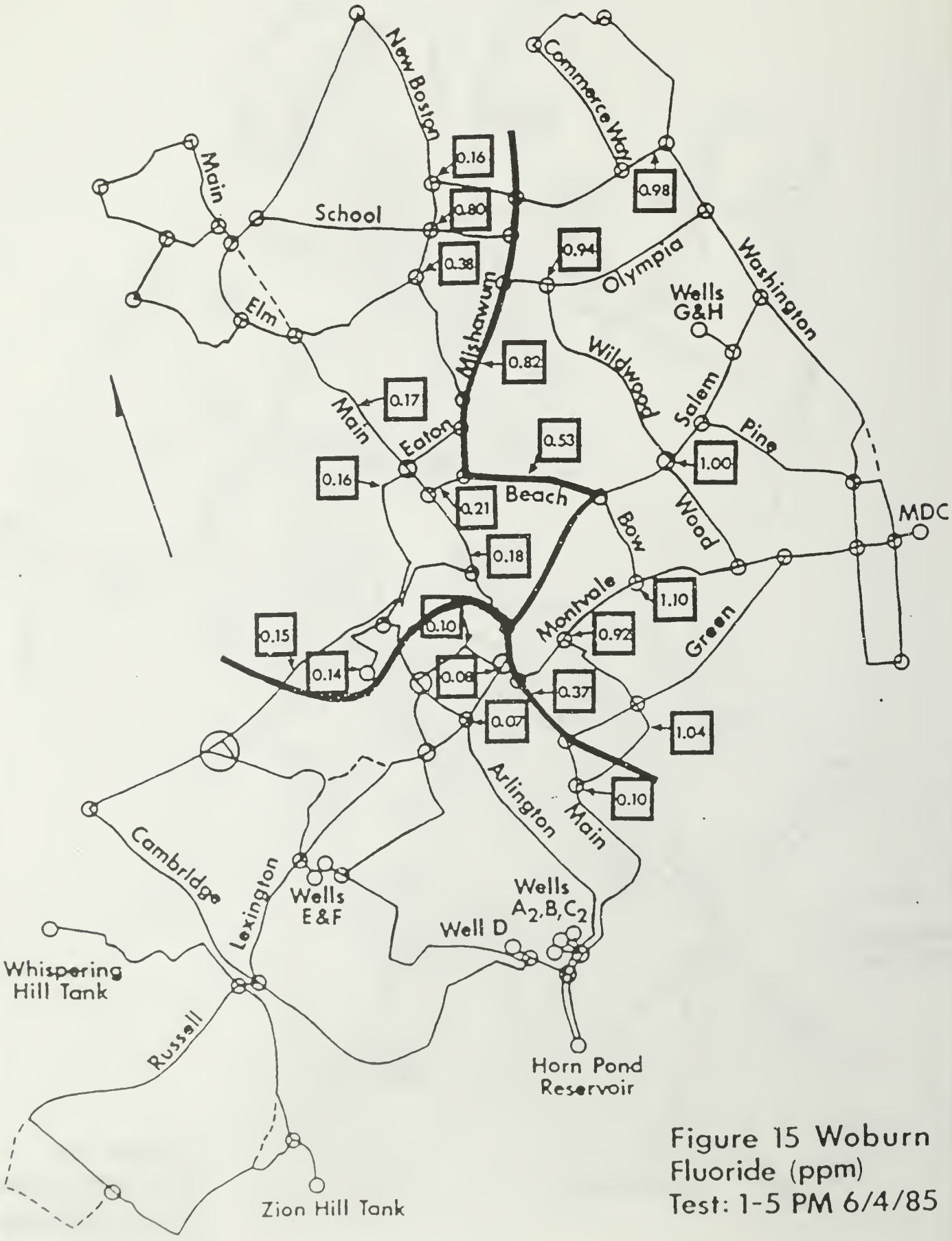

Figure 15 Woburn
Fluoride (ppm)
Test: 1-5 PM 6/4/85

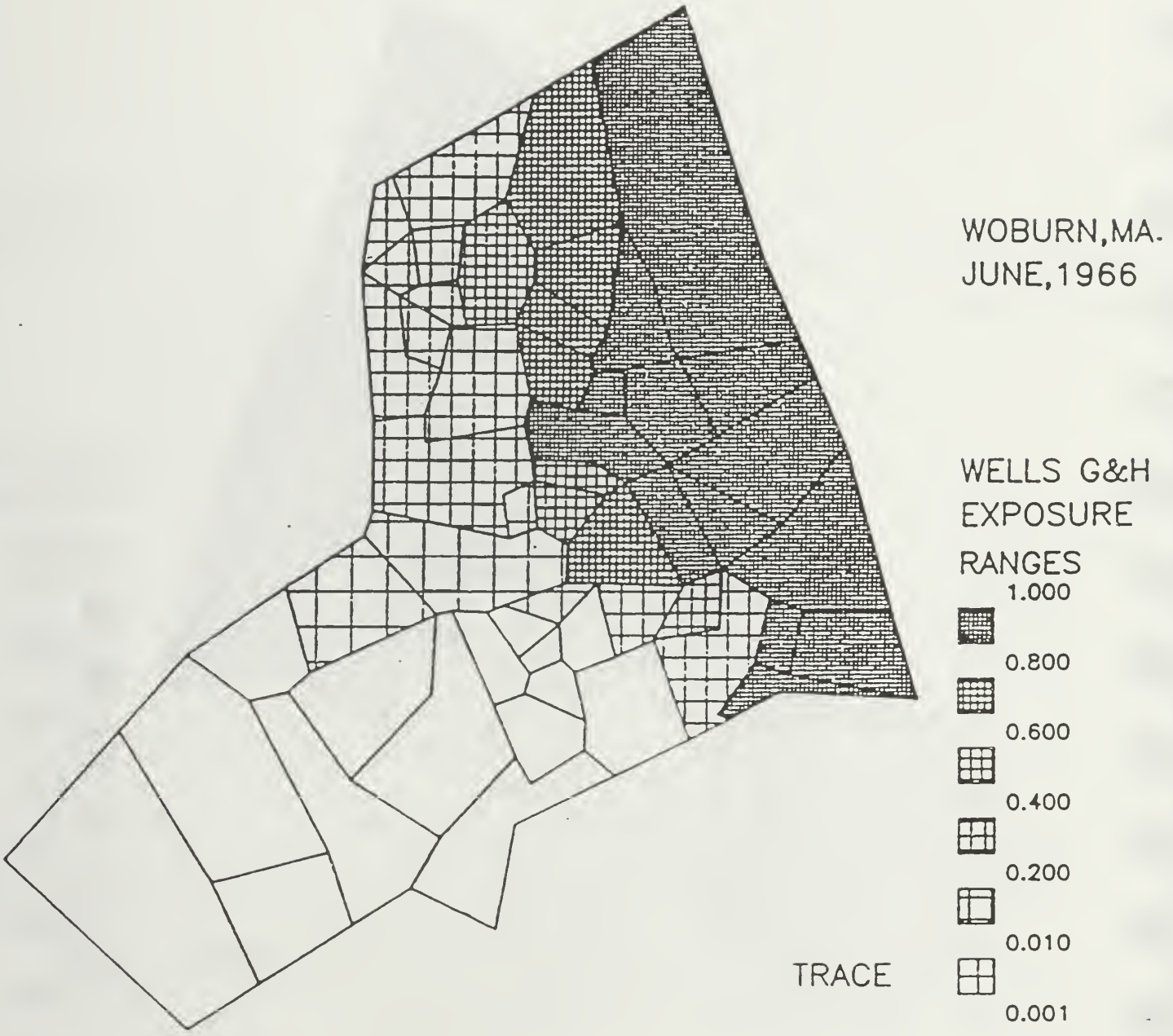

FIGURE 16. TYPICAL MEDIUM EXPOSURE AREA FOR 1964–69

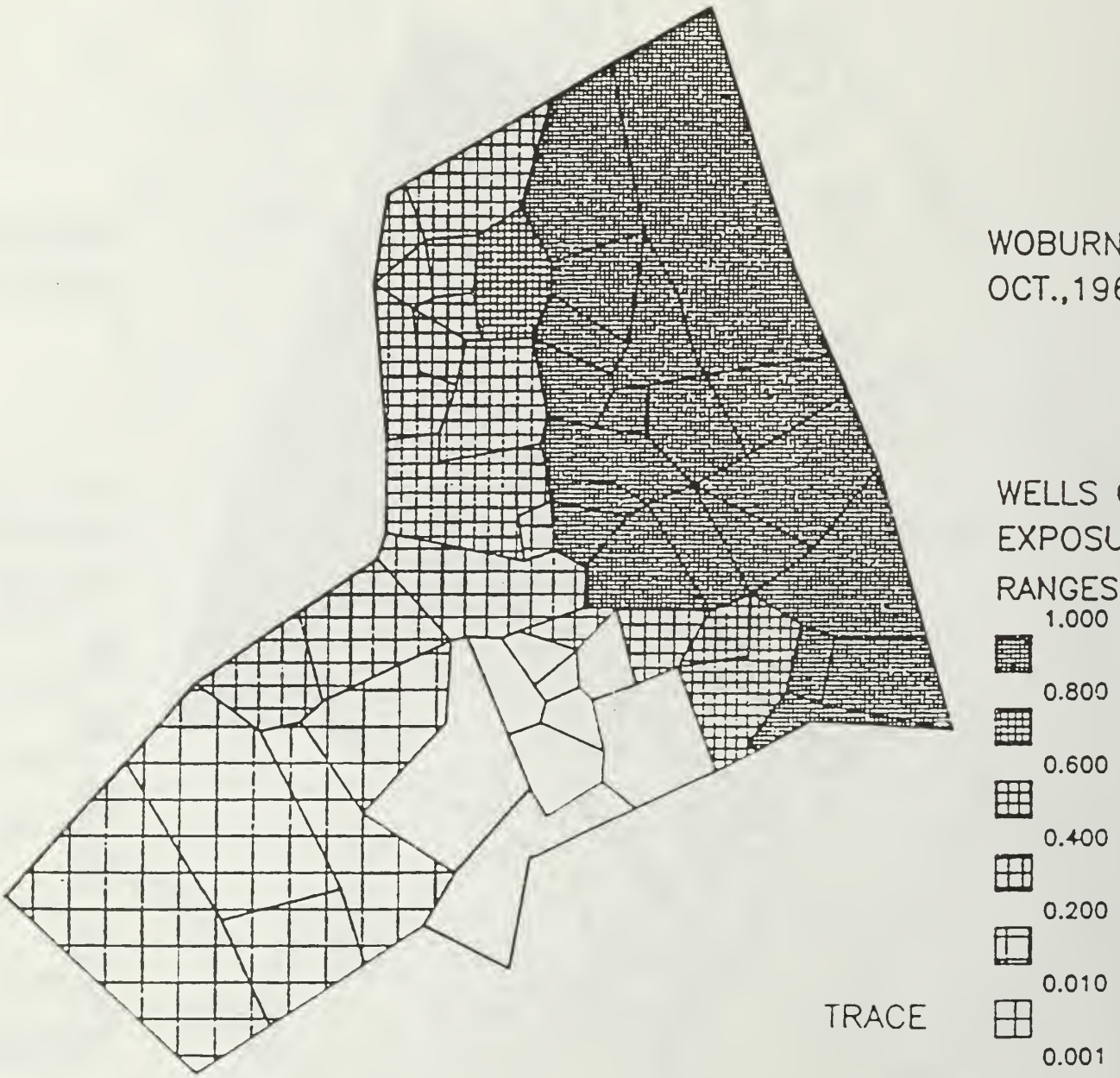

FIGURE 17. TYPICAL LARGE EXPOSURE AREA FOR 1964–69

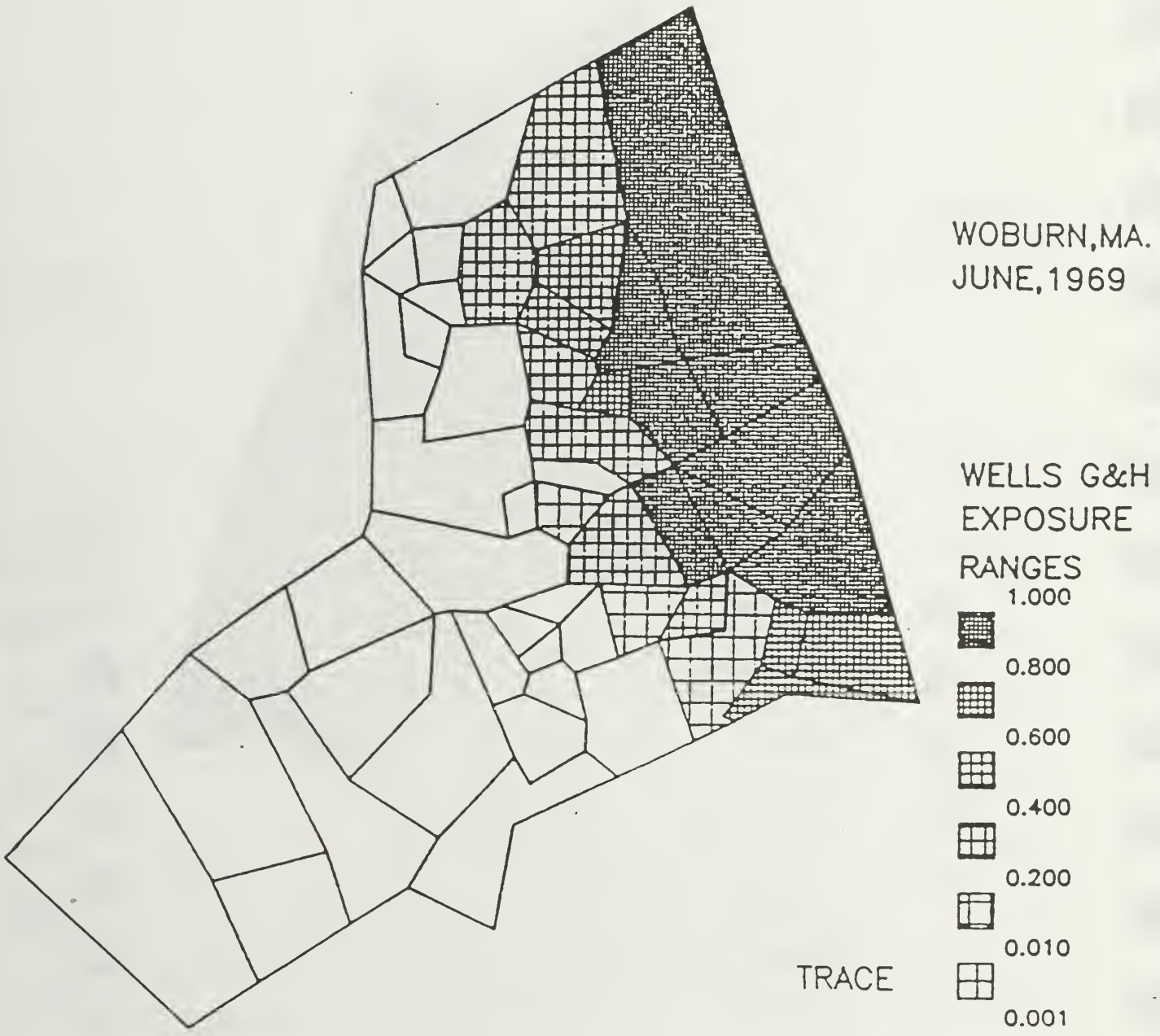

FIGURE 18. TYPICAL SMALL EXPOSURE AREA FOR 1964–69

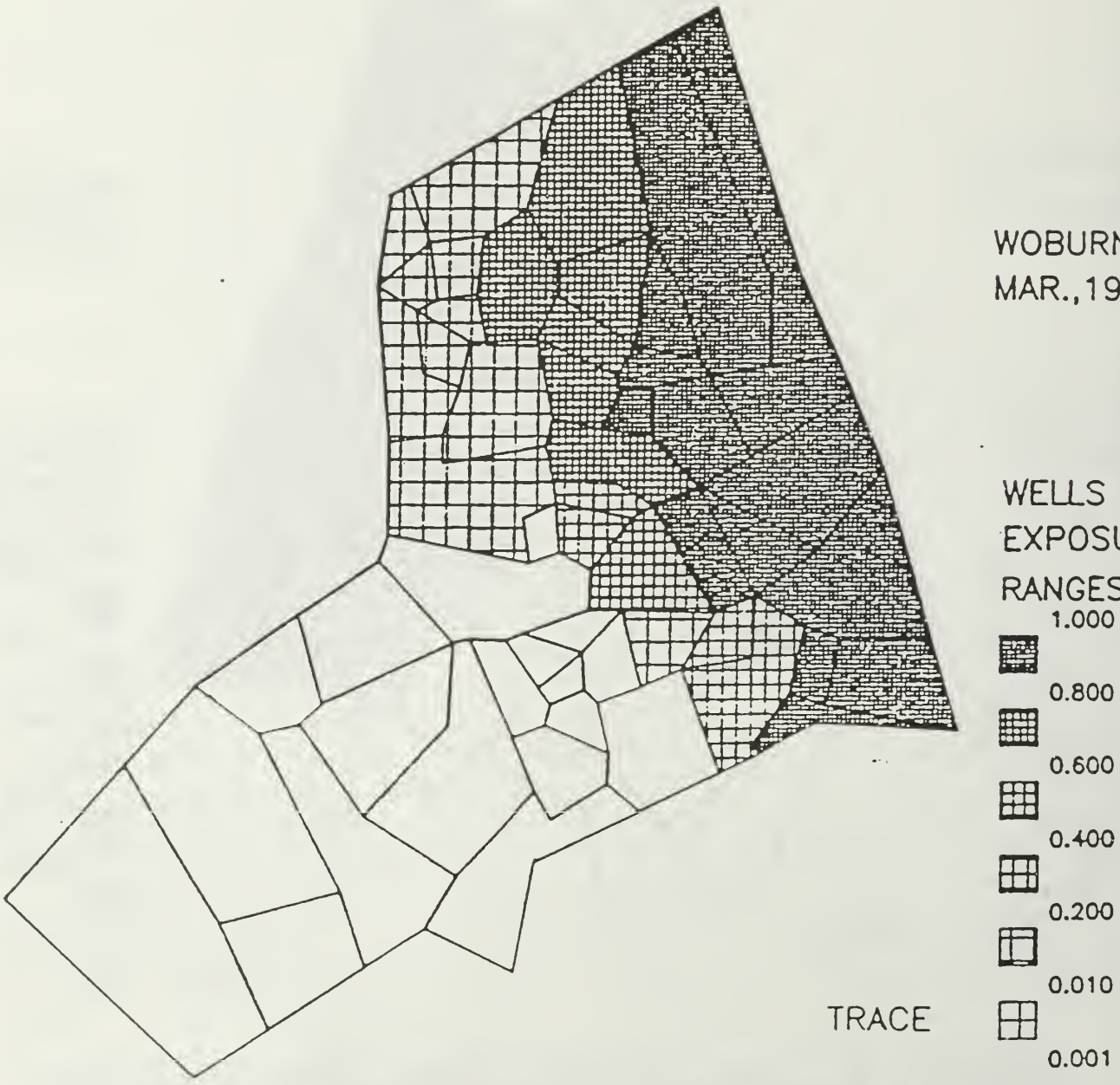

FIGURE 19. TYPICAL MEDIUM EXPOSURE AREA FOR 1970–79

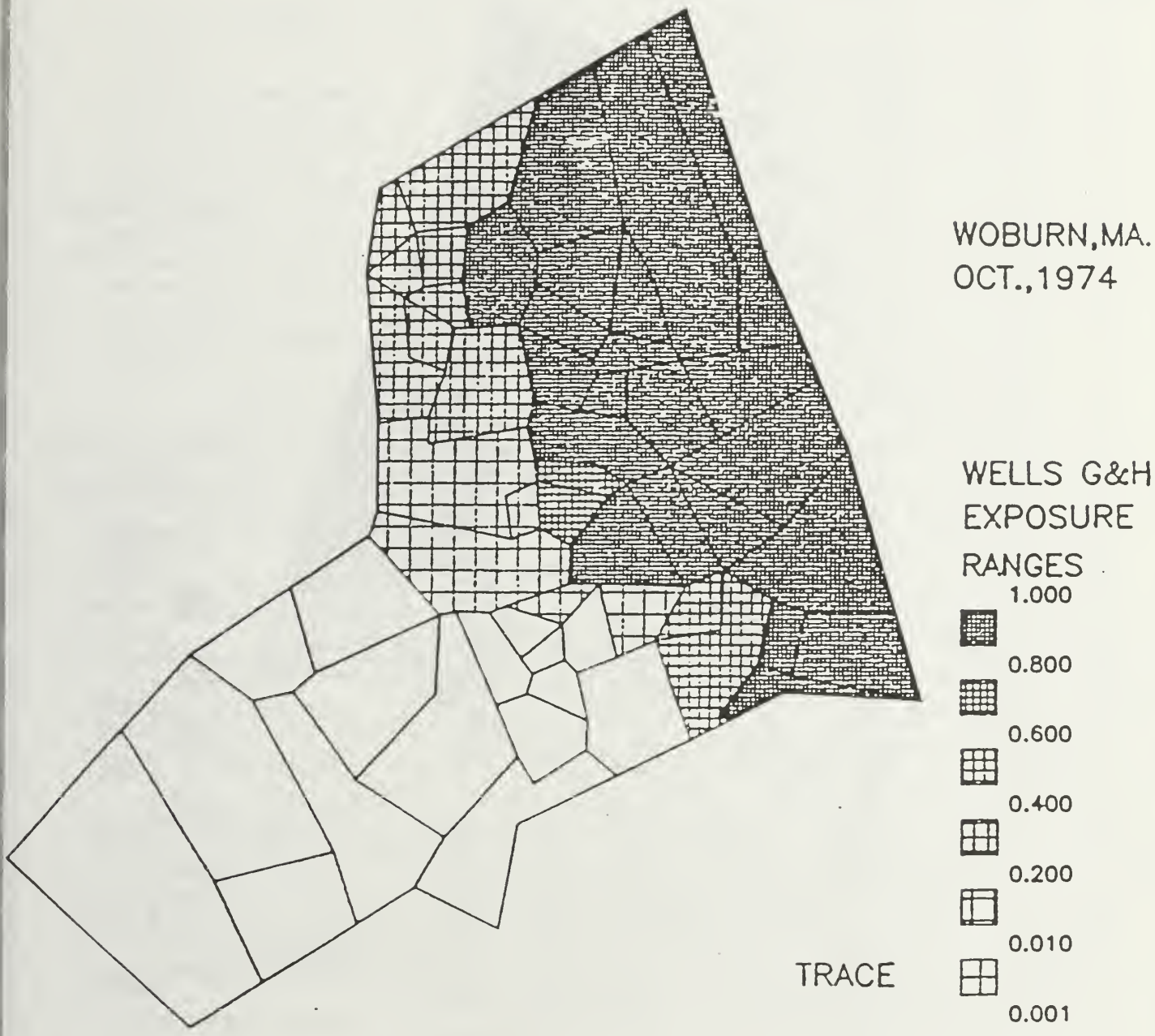

FIGURE 20. TYPICAL LARGE EXPOSURE AREA FOR 1970–79

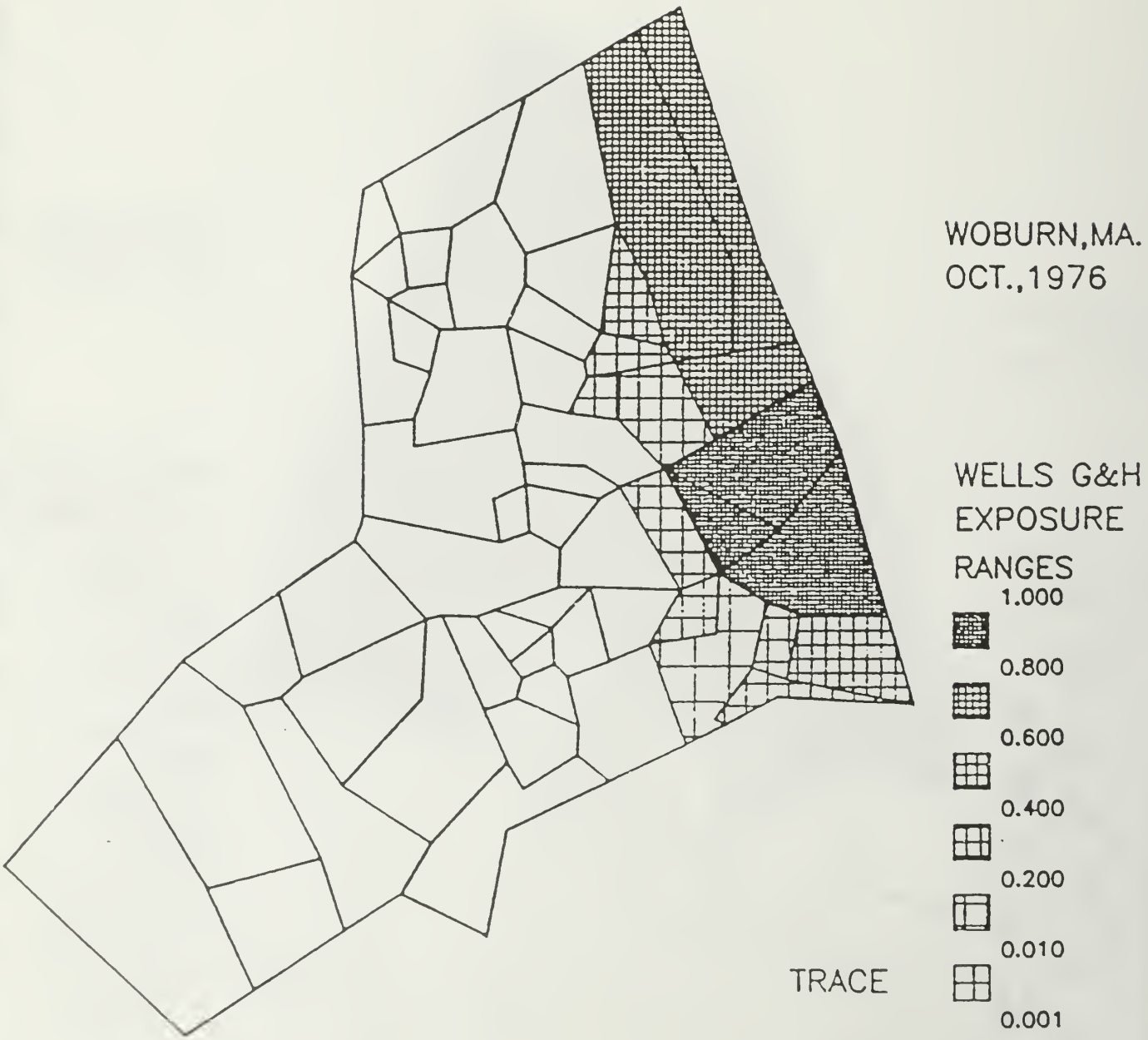

FIGURE 21. TYPICAL SMALL EXPOSURE AREA FOR 1970–79

Table 1. 1985 Principal Water Pipes in Woburn, Massachusetts

Street(s)	Length (ft.)	Diam. (in.)	Type*	Date Built	Age in 1985 (yrs.)	Pipe No.	From Node	To Node
Russell and Liana	4200	6	CI	1915	70	1	4	1
Lexington, Maura and Samoset	5200	10	CI	1963	22	2	3	1
Lexington & Waltham	3700	12	CI	1959	26	3	4	3
Waltham and Zion Hill	700	10	CI	1959	26	4	3	2
Cambridge, Revere, and Whispering Hill	4600	12	AC	1962	23	5	4	5
Booster Pump at Cambridge and Russell	400	12	CI	1959	26	6	6	4
Cambridge from Lexington to Bedford	4800	6	CI	1921	64	7	7	6
Bedford from Cambridge to So. Bedford	2800	10	CI	1921	64	8	8	7
Lexington from Cambridge to Well E	3000	6	CI	1913	72	9	9	6
Garden and South Bedford	4500	12	CI	1919/38	66/47	10	9	8
Well E connector	800	12	CI	1937	48	11	10	9
Lexington from Well E to Woburn Pkwy.	2600	6	CI	1913	72	12	9	11
Bedford and Kilby	7000	10	CI	1928	57	13	24	8
Cambridge from Russell to Well D	7200	12	CI	1959	26	14	12	6
Well D to Well F	5700	16	CI	1959	26	15	12	13
Water to connector to Well F	3700	16	CI	1909	26	16	13	11
Connector to Well F	100	10	CI	1959	26	17	14	13
Well D to Well C	800	14	CI	1900	85	18	12	15
Well C to Horn Pond Reservoir	500	16	CI	1900	85	19	15	16
Well C to Wells A_2,B,C_2	200	16	CI	1900	85	20	15	17
Lake and Arlington	6000	16	CI	1921	64	21	17	18
Pleasant from Woburn Pkwy. to N. Warren	1300	16	CI	1916	69	22	11	19
Lake and Main to Fowle	4700	16	CI	1913	72	23	17	19
Main from Fowle to Green	900	16	CI	1914	71	24	19	20
Main from Green to Montvale	2000	16	CI	1914	71	25	20	21
Main from Montvale to Pleasant	300	16	CI	1914	71	26	21	22
Pleasant from N. Warren to Main	1300	16	CI	1916	69	27	18	22
Main from Montvale to Salem	900	16	CI	1914	71	28	22	23
Main from Salem to Kilby	1400	16	CI	1915	70	29	23	24
Main from Kilby to Clinton	1900	16	CI	1917	68	30	24	25
Main from Clinton to Wyman	700	16	CI	1917	68	31	25	26
Ellis	1700	16	CI	1959	26	32	11	27

* CI = Cast Iron
AC = Asbestos Cement

N.Warren from Pleasant to Ellis	1100	10	CI	1910	75	33	18
Harrison and Winn to Pleasant	2100	16	CI	1959	26	34	22
N.Warren and Winn to Bedford	1750	20	CI	1959	26	35	27
Bedford and Hillside	1400	24	CI	1959	26	36	28
Kilby, Hart, and Wyman	3800	16	CI	1959	26	37	28
Fowle and Garfield to Green	1500	8	CI	1915/27	70/58	38	19
Green from Main to Garfield	1700	10	CI	1929	56	39	20
Montvale from Main to Auburn	1100	12	CI	1922	63	40	21
Auburn and Garfield to Green	3400	8	CI	1927	58	41	30
Montvale from Auburn to Bow	1900	12	CI	1922	63	42	31
Montvale from Bow to Wood	2200	12	CI	1922	63	43	33
Montvale from Wood to Green	1000	12	CI	1910	75	44	34
Green from Garfield to Montvale	4500	10	CI	1929	56	45	34
Montvale from Green to Central	1500	12	CI	1910	75	46	35
Montvale from Central to Washington	600	12	CI	1910	75	47	36
Montvale from Washington to MDC	100	12	CI	1975	10	48	37
Central and D Streets	3300	6	CI	1914	71	49	35
Washington from Montvale to D	2500	6	CI	1920	65	50	36
Washington from Montvale to Pine	1800	6	CI	1932	53	51	36
Central from Montvale to Pine	1300	12	CI	1975	10	52	35
Salem from Main to Bow	3400	10	CI	1920	65	53	23
Bow	2000	6	CI	1921	64	54	32
Salem from Bow to Wood	1500	10	CI	1920	65	55	40
Wood	2500	6	CI	1920	65	56	33
Salem from Wood to Pine	1200	10	CI	1920	65	57	42
Pine	3500	10	AC	1930	55	58	39
Washington from Pine to Cedar	3400	6	CI	1930	55	59	39
Salem from Pine to Wells G & H	1700	12	AC	1965	20	60	42
Salem and Cedar to Washington	1400	12	AC	1965	20	61	43
Wells G & H Connector	750	12	AC	1965	20	62	45
Clinton	800	6	CI	1928	57	63	25
Beach	3000	6	CI	1928	57	64	40
Eaton	1400	16	CI	1960	25	65	26

Mishawum from Clinton to Eaton	600	6	CI	1957	28	66	46	47
Mishawum from Eaton to Forest Pk.	600	16	CI	1960	25	67	47	48
Mishawum from Forest Pk. to Olympia	2500	16	CI	1960	25	68	48	49
Olympia from Mishawum to Wildwood	1000	16	CI	1960	25	69	49	50
Wildwood	5000	12	CI	1970	15	70	41	50
Olympia from Wildwood to Washington	3800	16	CI	1960	25	71	50	51
Washington from Cedar to Olympia	2000	12	AC	1965	20	72	44	51
Forest Pk. from Mishawum to Alfred	2800	16	AC	1970	15	73	48	52
Forest Pk. from Alfred to School	1100	16	AC	1970	15	74	52	53
School from Forest Pk. to Mishawum	1600	6	CI	1932	53	75	53	54
Mishawum from Olympia to School	1000	6	CI	1899	86	76	49	54
New Boston from School to New Industrial	1100	16	AC	1970	15	77	53	55
New Industrial	1800	12	AC	1971	14	78	55	56
Ryan	800	12	AC	1971	14	79	54	56
Mishawum from Ryan to Commerce	2500	16	CI	1978	7	80	56	57
Mishawum from Commerce to Washington	1100	24	CI	1973	12	81	58	57
Washington from Olympia to Mishawum	1600	16	CI	1973	12	82	51	58
Commerce Way	3200	20	CI	1973	12	83	57	59
Commonwealth Avenue	3700	10	AC	1977	8	84	59	60
Washington from Mishawum to Commonwealth	3300	6	CI	1920/38	65/47	85	58	60
New Boston from New Industrial to Merrimac	4100	16	AC	1970	15	86	55	61
Main from Wyman to Alfred	4100	16	CI	1917	68	87	26	62
Alfred	2900	6	CI	1931	54	88	52	62
Elm from Main to West	1300	12	CI	1920	65	89	62	63
Elm from West to Main	1800	12	CI	1920	65	90	63	64
Main from Elm to Nichols	500	12	CI	1927	58	91	64	65
School from Main to Merrimac	700	6	CI	1924	61	92	64	66
School from Merrimac to New Boston	3700	6	CI	1932	53	93	53	66
Merrimac	4900	6	CI	1924	61	94	66	61
Nichols	1200	12	CI	1927	58	95	65	67
Webster	1500	12	CI	1927	58	96	67	68
Pearl and West	3000	6	CI	1916	69	97	63	68

Pool and Winter	2000	6	CI	1925	60	98	67	6
Mountain	2200	6	CI	1923	62	99	70	69
Main from Nichols to Mountain	2000	6	CI	1913	72	100	65	70

*CI - Cast Iron
AC - Asbestos Cement

TABLE 2. 1984 USER DEMAND AREAS, BASED ON WATER METER ADDRESSES AND THE STREET INDEX FOR POLLING PLACES

Node Number	Addresses	Number of Residences
1	Anna Rd.	30
	Canterbury Rd.	19
	Carroll Rd.	28
	Carson Rd.	16
	Cerqua St.	10
	Dix Rd.	37
	Dix Rd. Ext.	
	Douglas Green	
	Duren Ave.	45
	Exeter Dr.	13
	Fortune Rd.	16
	Gayle St.	12
	Gettysburg St.	16
	Grant St.	10
	Hallmark Dr.	19
	Hampshire Cr.	3
	Helen Dr.	2
	Howard Ct.	6
	Johnson's Grant	
	Kendall's Mill	
	Kensington Ave.	10
	Kensington Dr.	
	Liana St.	19
	Linda St.	5
	Lee Rd.	18
	Lexington St., 310 to end	35
	Manomet Rd.	13
	Melo Rd.	11
	Michael's Green	
	Parliament Lane	22
	Penny Rd.	11
	Quail Run	
	Rich Rd.	27
	Robinson Rd.	50
	Roman Rd.	20
	Russell Ct.	
	Russell St., 206 to end	56
	Ryder Dr.	2
	Sachem Rd.	8
	Samoset Rd.	45
	Sawmill Brook Way	
	Saw Mill Brook Way	
	Senator Rd.	17
	Seneca Rd.	18
	Squanto Rd.	20
	Stonewall Dr.	11
	Strawberry Lane	3
	Tanners Circle	4
	Todd Rd.	10
	Vinebrook Way	

Node Number	Addresses	Number of Residences
3	Arcadia St.	7
	Aspen St.	6
	Aspin St.	
	Bernard Rd.	25
	Clifton St.	
	Glade St.	7
	Glenwood Ave.	27
	Grace Rd.	28
	Henderson Rd.	18
	Heritage Dr.	23
	Janis Dr.	
	Janis Tr.	8
	Jean Rd.	5
	Lexington St., 242 to 309	29
	Maura Dr.	21
	Minchin Dr.	26
	Morningside Cir.	
	Morningside Dr.	
	Morning Side Dr.	8
	Morning Side Circle	6
	New Village Rd.	1
	Parker St.	16
	Ridgewood Lane	7
	Summit St.	5
	Waltham St.	43
	Waverly Rd.	31
4	Adams Circle	5
	Battle March Way	
	Bay Rd.	
	Bay St.	6
	Belford Circle	11
	Briscoe Rd.	
	Briscoe St.	
	Brisco Rd.	15
	Brisco St.	
	Brook Rd.	4
	Crescent Rd.	13
	Dawes Circle	9
	Freedom Rd.	10
	Gilda Circle	
	Great Rd.	
	Grozier Cr.	
	Independence Dr.	59
	Kosciusko St.	10
	Lexington St., 200 to 241	7
	Matthew Place	
	Minuteman Rd.	
	October Ln.	
	Old Cambridge Rd.	2

Node Number	Addresses	Number of Residences
	Old Lexington Rd.	
	Old Lexington St.	
	Otis St.	12
	Patriot Cir.	
	Patriot Rd.	5
	Pheasant Ln.	12
	Prescott Way	7
	Revere Rd.	64
	Ridge Rd.	
	Russell St., 1 to 205	35
	Silver Mine Rd.	
	Silvermine Rd.	10
	Stevin Dr.	4
	Sylvanus Wood Ln.	34
	The Great Rd.	
	Tory Row	
	Whispering Hill Rd.	14
	Windsor Circle	
	Windsor Drive	
6	Cambridge Rd., 60 to end	37
	Chapel Way	
	Country Club Rd.	48
	Crossman Rd.	6
	Crawford Dr.	9
	Day Circle	53
	Flint Circle	
	Gately Drive	
	Glen Rd.	4
	Hiawatha Rd.	39
	Indian Hill Rd.	30
	Larch Rd.	4
	Ledgewood Rd.	9
	Lexington St., 145 to 199	10
	Locust St., 101 to end	
	Lover Lane	
	Loves Lane	5
	Mayflower Rd.	35
	Pond Terrace	9
	Rehabilitation Rd.	101
	Surrey Circle	4
	Surrey Rd.	33
7	Bedford Rd., 162-end even, 177-end odd	198
	Cambridge Rd., 1 to 59	130
	Churchill Rd.	14
	Downs Ct.	
	Kennedy Rd.	12
	Marlboro Rd.	63
	Princeton Rd.	6

Node Number	Addresses	Number of Residences
8	Banner Dr.	3
	Bamberg Dr.	6
	Bedford Rd., 34-160 even, 35-175 odd	46
	Bedford Tr.	2
	Bonnie Way	
	Brandt Dr.	16
	Bruce Rd.	11
	Burlington St., 71 to end	
	Cassidy Dr.	10
	Doherty Pl.	3
	Elijah St.	22
	Houghton St., 1 to 30	
	Jaycin Circle	5
	Katie Lane	
	Morrow Dr.	8
	Oak Knoll Circle	
	Oak Knoll Drive	
	Quimby Ave.	32
	Quimby Ave. Ext.	
	Rag Rock Dr.	16
	Rock St.	14
	Sheridan St.	36
	Sheilds St.	
	Shields St.	10
	South Bedford St.	23
	Thistle Rd., 18 to end (Burl. gap)	8
	Thornton St.	13
	Willow St., 48 to end	
9	Akerson Rd.	9
	Akeson Rd.	
	Acorn St.	4
	Brookland Rd.	3
	Bruno Tr.	10
	Burlington St., 1 to 70	
	Cannon Rd.	3
	Clifford Tr.	6
	Columbus Rd.	24
	Foley Rd.	3
	Gangi Tr.	5
	Garden Heights Ave.	7
	Garden Dr.	
	Garden St.	24
	Harrison Ave., 80 to end	21
	Houghton St., 31 to end	
	Kennedy Pk.	13
	Laurence Rd.	7
	Lexington St., 1 to 144	72
	Locust Cir.	
	Locust St., 1 to 100	24
	Mawn Ave.	

Node Number	Addresses	Number of Residences
	Mawn Dr.	2
	Mount Ida St.	8
	Totman Dr.	89
	Totman Rd.	
	Willow St., 1 to 47	
11	Adams Dr.	4
	Arlington Rd., 40-82 even, 37-81 odd	34
	Bacon St.	6
	Beacon St.	49
	Brandon Ct.	
	Ellis St., 1 to 37	18
	Evans Rd.	
	Evans St.	
	Geary Dr.	2
	Harrison Ave., 50 to 79	17
	Millet St.	1
	Millett St.	
	Oak St.	5
	Pleasant St., 94-end even, 99-end odd	45
	Reed St.	10
	Sturgis St., 38-end even, 35-end odd	14
	Thompson St.	6
	Water St.	27
	Woburn Parkway	9
	Wolcott Rd.	12
	Valley Rd.	23
17	Arlington Rd., 83 to end	19
	Barton Ln.	
	Border St.	12
	Buckman Ct.	
	Buckman St.	54
	Cove St.	
	Cranes Ct.	2
	Cranes Ln.	
	Cross St.	3
	Fays Lane	
	Lake Ave.	66
	Lake Circle	
	Lake Terr.	
	Lydon Ct.	5
	Lynden Ct.	
	Main St., 2-94 even, 1-107 odd	9
	Maria Ct.	5
	Pickering St.	11
	Stanley Tr.	6
	Stoddard St.	19
	Vining Ct.	10
	Wiley St.	10

Node Number	Addresses	Number of Residences
18	Arlington Rd., 2-38 even, 1-35 odd	114
	Bennett St.	23
	Caulfield Rd., 15 to end	6
	Church Ave., 15 to end	8
	Converse Pl.	
	Court St.	29
	Ellis St., 38 to end	9
	Foster St.	11
	Grove St.	11
	Harrison Ave., 15 to 49	12
	Hillcrest Rd.	2
	Morse St.	
	Munroe St.	17
	North Warren St., 1 to 60	36
	North Warren Street Ext.	
	Pleasant St., 44-92 even, 45-97 odd	41
	Wade Place	48
	Warren Ave., 2-38 even, 1-29 odd	27
19	Ash St.	12
	Brooks St.	4
	Buck Ct.	
	Buck St.	12
	Conn St.	44
	Conn Ct.	
	Dows Ln.	
	Dow's Ln.	8
	Fowle St., 2-62 even, 1-21 odd	25
	Hudson Pl.	
	Hudson St.	32
	Innitou Rd.	13
	Innitou Rd. Ext.	
	John St.	15
	Lakeview Tr.	20
	Lawrence St.	24
	Main St., 96-200 even, 107A-201 odd	10
	Pierce St.	3
	Porter St.	51
	Richardson St.	41
	Richardson Street Ext.	
	Richmond Pk.	14
	Sturgis St., 2-36 even, 1-33 odd	37
	Veteran Rd.	11
	Warren Ave., 40-end even, 31-end odd	4
	Warren Rd.	4
20	Edgehill Rd.	20
	Green St., 1 to 17	4
	Highland St., 2-14 even, 1-19 odd	18
	Main St., 202 to 280	59
	Miller Pl.	

Node Number	Addresses	Number of Residences
	Mt. Pleasant St., 1 to 10	13
	Myrtle St.	10
	Prospect St., 30 to end	41
	Summer St.	10
21	Buel Pl.	
	Caulfield Rd., 1 to 14	6
	Church Ave., 1 to 14	5
	Common St., 1 to 6	
	Everett St., 31 to end	7
	Gallagher Ave.	
	High St., 1 to 40	6
	Main St., 281 to 375	9
	Montvale Ave., 2-50 even, 1-31 odd	4
	Prospect Ave.	8
	Prospect St., 1 to 29	16
	Walnut St., 21 to end	9
22	Abbott St.	
	Common St., 7 to end	
	Everett St., 1 to 30	12
	Federal St.	11
	Harrison Ave., 1 to 14	3
	Main St., 376 to 438	1
	Park St., 1 to 23	
	Pleasant St., 1 to 43	
	Walnut St., 1 to 20	9
	Winn St., 1 to 79	38
23	Campbell St., 1 to 39	16
	Center St.	34
	Centre St.	
	Ellis Ct.	11
	Franklin St.	37
	Harlow Ct.	7
	Hovey Ct.	
	Hovey St.	13
	Hubbard Pl.	
	Manns Ct.	10
	Mann's Ct.	
	Main St., 439 to 524	7
	Park St., 24 to end	18
	Salem St., 1 to 75	58
	Spring St.	8
	Union St., 1 to 31	21
	Wade Ave., 1 to 12	48
	Winn Pk.	37
24	Bedford Rd., 1 to 33	
	Carter Place	1
	Charles St.	15

Node Number	Addresses	Number of Residences
	Chesnut St.	
	Chestnut St.	49
	Church Ct.	4
	Church St.	24
	Colonial Rd.	23
	Cummings Ave.	
	David Circle	6
	Davis St.	37
	Flagg St.	17
	Frances St.	29
	Francis St.	
	Hillside Ave.	
	James St.	20
	Johnson St.	7
	Kilby Dr.	4
	Kilby St.	115
	Lantern Ln.	3
	Linden Circle	3
	Linden St.	11
	Linden Street Ext.	
	Lisa Dr.	2
	Main St., 526-616 even, 525-607 odd	46
	Manning St.	12
	Middlesex St.	9
	Mishawum Rd., 2-10 even, 1-13 odd	9
	North Warren St., 61 to end	72
	Plympton St.	23
	Scott St.	28
	Thistle Rd., 1 to 5 (Burl. gap)	5
	Village St.	
	Winn Ct.	2
	Winn St., 80 to end	78
	Wright St.	25
25	Blake St.	2
	Blake Terr.	6
	Boys Club Lane	
	Clinton St., 1 to 15	10
	Main St., 618-670 even, 609-671 odd	28
	Page Place	10
	Place Ln.	4
26	Anderson Way	
	Biltmore St.	
	Brae Circle	26
	Brentwood Rd., 1 to 26	23
	Brown Place	11
	Colony Rd.	6
	Coolidge Rd.	4
	Curtis Rd.	10
	Dickson Rd.	7

Node Number	Addresses	Number of Residences
	Eaton Ave., 12-30 even, 11-43 odd	20
	Eaton Lane	
	Ellen Rd.	15
	Elmwood Pl.	
	Elmwood Tr.	2
	Frances Rd.	29
	Frederick Cir.	13
	Frederick Dr.	
	Glendale Ave.	
	Hamilton Rd.	28
	Hart Place	24
	Hart St.	41
	Intervale St.	11
	Jericho Rd.	5
	Kennere Ave.	
	Kimball Rd.	13
	Lowell St.	58
	Magnolia Ave.	
	Main St., 672 to 732	30
	Marion Ave.	14
	Meadowbrook Ln.	
	Meadow Lane	
	Millyan Rd.	9
	Murray Rd.	5
	Murray Road Ext.	
	Newbury St.	14
	Park Dr.	38
	Park Drive Ext.	5
	Pilgrim Rd.	16
	Sendick Rd.	13
	Sheila Ave.	10
	Wilcox Circle	
	Wildmere Ave.	
	Wyman St.	51
30	Abbott Ct.	3
	Abbott Pl.	
	Allen St.	11
	Arlington St.	27
	Belmont St.	13
	Blueberry Hill Rd., 1 to 38	43
	Blossom St.	4
	Bradford Rd.	16
	Bryant St.	13
	Carter St.	20
	Clark St.	5
	Cliffside Tr.	2
	Concord St.	
	Crest Ave.	
	Eastern Ave., 51 to end	56
	Fowle St., 64-end even, 23-end odd	18

Node Number	Addresses	Number of Residences
	Fulton St.	20
	Garfield Ave., 34-end even, 27-end odd	60
	Glenwood St.	13
	Golden Terrace	2
	Green St., 18-104 even, 19-91 odd	49
	Highland Ave.	5
	Highland St., 16-end even, 21-end odd	27
	Jefferson Ct.	6
	Leonard St.	32
	Madison St.	13
	Manning Ct.	
	McCabe Ct.	3
	Medford St.	
	Mount Pleasant Ct.	
	Mount Pleasant St., 11 to end	60
	Oxford Place	4
	Playstead Ave.	7
	Skyview Ln.	8
	Skyview Rd.	6
	Sonar Dr.	
	Sonrel St.	18
	South St.	13
	Spring Ct.	127
	Spring Court Ext.	
	Spring Court Terr.	
	Stoneham St.	
31	Auburn St.	49
	Campbell St., 40 to end	132
	Fairmount St.	26
	First St.	7
	Garfield Ave., 2-32 even, 1-25 odd	18
	Greenwood Ave.	10
	Hanson Ct.	5
	High St., 41 to end	29
	Jefferson Ave.	7
	Montvale Ave., 52-88 even, 33-87 odd	79
	Second St.	3
	Union St., 32 to end	21
	Wade Ave., 13 to end	48
32	Boline Pl.	16
	Bow St., 1 to 32	15
	Cook Terr.	
	Eastern Ave., 1 to 50	30
	Ferguson Pl.	5
	Gardner Ave.	8
	Holden Pl.	8
	Ingalls St.	11
	Liberty Ave.	48
	Montvale Ave., 89 to 159	54

Node Number	Addresses	Number of Residences
	Montvale Ln.	8
	Oakwood Ln.	3
	Sherman Place	35
	Sherman Place Ct.	
	Sherman Terr.	
	Sullivan Pl.	4
	Sullivan St.	
	Vernon St., 1 to 33	31
33	Argent St.	
	Chandler St.	
	Deb Rd.	9
	Harvard St.	16
	Harvard St. Ext.	
	Laurel St.	4
	Montvale Ave., 160 to 210	24
	Nashua St., 88 to end	14
	Packard St.	2
	Theresa Rd.	6
	Wood St., 43 to end	15
34	Arnold Rd.	
	Blueberry Hill Rd., 39 to end	
	Boston St.	
	Connors Dr.	
	Draper St.	1
	Eagle Rd.	9
	Fieldstone Dr.	16
	Fox Rd.	
	Frank St.	13
	Frank Street Ct.	
	Green St., 106-end even, 93-end odd	14
	Hawley St.	
	Hawk Rd.	4
	Henry Ave.	13
	Holton St.	11
	James Terr.	18
	May St.	
	Micro Dr.	
	Montvale Ave., 211 to 263	12
	Nashua St., 1 to 87	47
	Paul Ave.	5
	Pigeon Rd.	
	Tremont St.	30
	Vernon St., 34 to end	17
	Willard Rd.	
	Williams Ave.	22
	William Ave.	
	Woburn St.	
35	Asbury Ave.	12

Node Number	Addresses	Number of Residences
	Ashbury Ave.	
	Central Ct.	
	Central St., 22 to 76	17
	Eldridge St.	
	Erie St., 1 to 9	5
	Grape St., 1 to 16	6
	Orange St., 29 to end	23
	Montvale Ave., 264 to 285	6
	Silk St.	
	Utica St., 12 to end	4
36	A St.	
	Albany St.	14
	B St.	
	Erie St., 10 to end	7
	Fremont St.	6
	Grape St., 17 to end	14
	Hawthorne St., 1 to 11	4
	Henshaw St.	1
	Hill St.	1
	Mack Rd.	
	Maple St.	
	Martin St.	
	Merrill St.	
	Montvale Ave., 286 to end	9
	Sherman St.	
	Utica St., 1 to 11	6
	Washington St., 22 to 124	39
38	C St.	
	Cedar St.	
	Central St., 77 to end	10
	D St.	
	Middlesex Ave.	
	Ran Dr.	
	Washington Ave., 5	1
	Washington St., 1 to 21	7
39	Albert Dr.	60
	Carmen Tr.	16
	Carter Rd.	4
	Central St., 1 to 21	15
	Felton St.	22
	Floyd St.	11
	Gregg St.	19
	Hawthorne St., 12 to end	11
	Lakeview Ave.	
	Lawson St.	12
	Lincoln Dr.	
	Mill St.	213
	Mill Terr.	19

Node Number	Addresses	Number of Residences
	Munroe Ave.	14
	Montvale Rd., 66 to end	18
	My St.	
	Nason Terr.	
	Orange St., 1 to 28	18
	Perry St.	6
	Pine St., 67 to end	32
	Radcliffe Way	3
	Radclyffe Way	
	Salem St., 378 to end	10
	Smith Ln.	
	Unicorn Dr.	
	Washington Circle	20
	Washington St., 125 to 272	30
	Washington Tr.	6
	Wilbur St.	
40	Alpena Ave.	7
	Beach St., 54 to end	16
	Bow St., 33 to end	15
	Buttaro Rd.	29
	Continental Ct.	72
	Creston Ave.	72
	Dearborn Tr.	3
	Field Tr.	4
	Hilltop Cr.	7
	Hilltop Parkway	17
	Hilltop Tr.	16
	Hyde Ave.	2
	Kathleen Dr.	
	Laura Rd.	
	Loker Lane	
	Maple Ave., 1 to 30	17
	Maple Ct., 1 to 30	
	Memorial Ave.	
	Parkview Rd.	12
	Roger Ave.	
	Rogers Ave.	5
	Salem St., 76 to 173	48
	Sedgewick Park	9
	Sedgewick Park Ext.	
	Walnut Ct.	9
	Westview Tr.	11
41	Albert St.	7
	Hancock St.	3
	Hinston Rd., 1 to 15	11
	Joshua St.	
	Lillian St., 2-32 even, 1-19 odd	21
	Maple Ave., 31 to end	12
	Maple Ct., 31 to end	

Node Number	Addresses	Number of Residences
	Salem St., 174 to 235	23
	Sunset Ave.	5
	Wildwood Ave., 1 to 275	
	Winstead Ave.	17
	Wood St., 1 to 42	13
42	Bird St.	11
	Chase St.	9
	Crescent Ave.	3
	Dale St.	11
	Elm Ave.	19
	Lincoln Rd.	9
	Montvale Rd., 1 to 65	35
	Pine St., 1 to 66	19
	Riley Rd.	
	Salem St., 236 to 280	6
	Walnut Hill Park	
	Walnut Hill St.	
44	Aberjona Dr.	
	Carlena Tr.	11
	Cedar Dr.	
	Cedar St.	5
	Forbes Rd.	
	Forbes St.	
	George Ave.	13
	Lynn St.	5
	Marilyn Ct.	7
	Pento Rd.	
	Rifle Range Rd.	2
	Robert Ave.	14
	Salem Ave.	12
	Salem St., 281 to 377	24
	Schneider Ct.	
	Spartan Circle	
	Stephanie Cr.	12
	Washington St., 273 to 368	18
46	Anthony Dr.	6
	Beach St., 1 to 53	29
	Beach Terr.	8
	Cleveland Ave.	29
	Clinton St., 16 to end	12
	Kendall St.	3
	Knollwood Ave.	7
	Maywood Ln.	
	Maywood Terr.	21
	Middle St., 1 to 9	8
	Mishawum Rd., 12-52 even, 15-51 odd	34
47	Dodge Rd.	1

Node Number	Addresses	Number of Residences
	Dorothy Dr.	3
	Eaton Ave., 32-end even, 45-end odd	5
	Fairview Rd.	14
	Fairview Terr.	8
	Fryberg Rd.	
	Fryburg Rd.	6
	Markham Ave.	
	Markham Terr.	
	Middle St., 10 to 63	12
	Mishawum Rd., 54-74 even, 53-77 odd	23
48	Atwood Ave.	1
	Birchwood Ln.	
	Brentwood Rd., 27 to end	14
	Carpenter Ct.	
	Cranston Cr.	7
	Edith Ave.	7
	Elaine Rd.	5
	Emeline St.	13
	Evangeline Ln.	8
	Forest Park Rd., 1 to 35	30
	Forest Park Cr.	4
	Hinston Rd., 16 to end	15
	Lillian St., 34-end even, 19A-end odd	11
	Majority Lane	
	Mishawum Rd., 76-130 even, 79-129 odd	18
	Middle St., 64 to end	26
	Overlook Ave.	3
	Park View Ave.	
	Phillips St.	12
	Red Leaf Ln.	1
	Sanborn Ave.	
	Susan Tr.	8
49	Hall St.	3
	Highet Ave.	21
	Mishawum Rd., 131 to 191	23
	Olympia Ave., 83 to end	1
	Pine Grove Ave.	5
	Rumford Park Ave., 18 to end	17
	University St.	1
50	Arrow Dr.	
	Hazel Ct.	
	Olympia Ave., 61 to 82	
	State St.	
	Wheeling Ave.	
	Wildwood Ave., 276 to end	
51	Dewey Ave.	18
	Hobson Ave.	2

Node Number	Addresses	Number of Residences
	Marietta St.	6
	Normac Rd.	
	Olympia Ave., 1 to 60	7
	Oregon Ave.	2
	Wainwright Ave.	2
	Washington St., 369 to 443	7
52	Alfred St., 24-end even, 27-end odd	16
	Barbara Circle	19
	Boyd Rd.	24
	Cronin Way	
	Forest Glen Circle	7
	Forest Glen Rd.	8
	Forest Park Rd., 36 to 95	14
	Rumford Park Ave., 1 to 17	11
	Thomas St.	12
53	Emerson Rd.	3
	Forest Park Rd., 96 to end	8
	Hope Ln.	8
	Karen Rd.	13
	Lucia Terr.	
	New Boston St., 1 to 133	
	School St., 80 to 203	38
	Woodside Terr.	22
54	Alice Rd.	5
	Allan St.	2
	Bronislaw St.	12
	Jan Rd.	
	Jan St.	12
	Old Mishawum Rd.	3
	School St., 204 to end	15
	Mishawum Rd., 192-230 even, 193-275 odd	4
55	Adele Rd.	
	Gill St.	
	Everberg Rd.	
	Industrial Pkwy., 16-end even, 31-end odd	
	Industrial Rd., 16-end even,31-end odd	
	New Boston St., 134 to 181	
	Roessler Rd.	
	Sixth Rd.	
	Torrice Dr., 1 to 19	
56	Industrial Pkwy., 2-14 even,1-29 odd	
	Industrial Rd., 2-14 even, 1-29 odd	
	Linscott Rd.	
	Mishawum Rd., 232-278 even, 277-279 odd	
	Rath Rd.	
	Ryan Rd.	

Node Number	Addresses	Number of Residences
57	Commerce Way, 1 to 36	
	Eastern Boul.	
	Elmwood St.	10
	Forest Ave.	
	Forest St.	1
	Garden Tr.	12
	Mishawum Rd., 280 to end	7
	Nelson St.	
58	Dragon Ct., 1 to 19	6
	Dragon Court Circle	6
	Richard Cr.	49
	Washington St., 444 to end	9
59	Atlantic Ave.	
AKA 57	Cabot Rd.	
	Commerce Way, 37 to end	
	Commonwealth Ave.	
	Constitution Ave.	
	Constitution Way	
	Puritan Ave East	
	West Congress Ave.	
60	Derry Ln.	
AKA 58	Dragon Ct., 20 to end	8
	Florence Terr.	2
	North Dragon Ct.	
61	Alger St.	
	Avon St.	
	Dundee Dr.	
	East Dexter Ave., 1, 101 to end	
	Eddy St.	
	Fifth Rd.	
	First Rd.	1
	Fourth Rd.	
	Hinckley St.	
	Knight Ave.	
	Knight St.	
	Linwood Ave.	
	Merrimac St., 76A-end	17
	Mishawum Ave.	
	Monks St.	
	Newberg Ave.	
	New Boston St., 182 to end	
	North Maple St. 3,9,17,18,20,22,22A	6
	Oakland St.	
	Palmer St.	
	Presidential Dr.	
	Presidential Rd.	

Node Number	Addresses	Number of Residences
	Rumford Ave.	
	Second Rd.	
	Suffolk St.	
	Tedesco Dr.	
	Temple St.	
	Third Rd.	9
	Torrice Dr., 20 to end	
	White St.	
	Undercover Way	
62	Alfred Pl.	
	Alfred St., 2-22 even, 1-25 odd	6
	Alfred Street Ext.	
	Alfred Terr.	6
	Baldwin Green	2
	Elm St., 1 to 29	9
	Fisher Tr.	25
	Fletcher Rd., 28 to end	26
	Giacalone Ln.	
	Giacalone Rd.	
	Harold Ave.	9
	Main St., 733 to 869	3
	Middlesex Canal Park	
	Rear Alfred St.	
	True Pl.	
	Van Norden Rd., 19 to end	15
63	Elm St., 30-72 even, 31-61 odd	24
	Newbridge Ave.	11
	Patricia Cr.	7
	Perry Pl.	5
	Tidd Ave.	6
	Traverse St., 10 to end	6
	Ward St.	22
	West St.	30
64	Elm St., 74-end even, 63-end odd	6
	Edwards Rd.	24
	Keith Cr.	16
	Main St., 870 to 901	3
	School St., 1 to 13	11
	Traverse St., 1 to 9	
65	App Ct.	6
	Dexter Ave., 1 to 13 (end)	12
	Donna Rd.	10
	Donna Road Ext.	
	East Nichols St.	14
	Lord Terr.	2
	Main St., 902-972 even, 903-949 odd	30
	Milan Ave., 33 to end	12
	Minot St.	7

Node Number	Addresses	Number of Residences
	Nichols St.	19
	O'Neil Rd.	2
	Pettiglio Terr.	2
	Rear Dexter Ave.	
	Wilson St.	
	Wyman Pl.	
66	Baldwin Ave.	2
	Baldwin St.	
	Banks St.	3
	Belmont Ave.	
	Chester Ave.	21
	Cottage St.	3
	Cutting Ave.	1
	Danforth Ave.	4
	Daniel Dr.	
	Dartmouth St.	12
	East Dexter Ave., 14 to 100	
	Eighth Rd.	1
	Fletcher Rd., 1 to 27	19
	Foster Ave.	7
	Gerard St.	
	Glen St.	
	Hensel Ave.	
	Hillner Ave.	
	Jones Ave.	8
	Longwood Ave.	8
	Merrimac St., 1 to 76	22
	Michael Cr.	34
	Mostika Rd.	4
	Niehouse Ave.	
	Niehouse St.	
	Poplar St.	7
	Prescott St.	
	School St., 14 to 79	48
	Tyler St.	
	Van Norden Rd., 1 to 18	10
	Williams Ct.	5
67	Gatta Cr.	13
	Poole St., 2-34 even, 1-39 odd	30
	Nichols St. Ext.	54
	Webster Ave.	60
	Webster Ave. Ext.	3
	Webster Ct.	
	Webster St., 1 to 19	1
68	Bartlett Dr.	26
	Dickie Rd.	9
	Florence Rd.	
	Kendal Dr.	

Node Number	Addresses	Number of Residences
	Kendall Dr.	17
	Kimball Ct.	1
	Pearl St.	292
	Spring Garden Tr.	4
	Sunnyside Rd.	
	Sylvan Rd.	2
	Webster St., 20 to end	11
	Westgate Dr.	
69	Briarwood Rd.	16
	Cedarwood Rd.	10
	Mountain St., 45 to end	19
	Norwood Cr.	
	Poole St., 36-end even, 41-end odd	9
	Robinlea Cr.	12
	Winter Rd.	49
	Winter St.	11
	Williams Ln.	6
70	Albion St.	
	Alpine Ave.	
	Ashburton Ave.	20
	Belle View Ave.	
	Breed Ave.	
	Dakota St.	
	Delaware Ave.	2
	Driftwood Dr.	
	Florence Ave.	
	Ford Tr.	2
	Glen Ave.	
	Grafton St.	
	Granville St.	
	Indiana Ave.	2
	Ingelow Ave.	
	Kearsage Ave.	6
	Kentucky Ave.	4
	Lamoille St.	
	Lamoil St.	6
	Lancaster St.	
	Lee St.	
	Lenox Ave.	
	Leslie Ave.	
	Louisiana Ave.	
	Main St., 974-end even, 951-end odd	145
	Maryland Ave.	
	Massachusetts Ave.	2
	Marian St.	2
	Milan Ave., 1 to 32	25
	Missouri Ave.	
	Montgomery Ave.	
	Mountain St., 1 to 44	17

Node Number	Addresses	Number of Residences
	Naples Ave.	10
	North Maple St., 4,5,6,10,11,15,16, 26,27,29,31,40	12
	North Massachusetts Ave.	
	North Washington Ave.	
	Oregon St.	
	Park Ave.	
	Pennsylvania Ave.	
	Richmond Ave.	11
	Sacremento Ave.	
	Spencer Ave.	
	Summit Ave.	
	Tennessee Ave.	1
	Tennessee St.	
	Thurman St.	
	Townsend St.	
	Virginia Ave.	2
	Wachusett Ave.	
	Washington Ave.	8
	Wheeling St.	3
	York St.	

TABLE 3. 1964-1969 USER DEMAND AREAS BY APPROXIMATE GEOCODE IN DIME
FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101
AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT
ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	101025	17270	1030424535	533846424612	533882	0	01
25	17270	101025	17270	1040424612	533882424727	533953	0	02
25	17270	101025	179999999990424727		533953424644	534026	0	03
25	17270	101025	179999999990424644		534026424629	534039	0	04
25	17270	101025	179999999990424629		534039424500	533899	0	05
25	17270	101025	179999999990424500		533899424535	533846	0	06
25	17270	103025	179999999990424535		533846424580	533777	0	07
25	17270	103025	17270	1060424580	533777424635	533795	0	08
25	17270	103025	17270	1040424635	533795424612	533882	0	09
25	17270	104025	17270	1060424635	533795424750	533855	0	10
25	17270	104025	17270	1070424750	533855424785	533902	0	11
25	17270	104025	179999999990424785		533902424727	533953	0	12
25	17270	106025	179999999990424580		533777424608	533734	0	13
25	17270	106025	17270	1170424608	533734424646	533712	0	14
25	17270	106025	17270	1110424646	533712424690	533779	0	15
25	17270	106025	17270	1090424690	533779424757	533826	0	16
25	17270	106025	17270	1070424757	533826424750	533855	0	17
25	17270	107025	17270	1090424757	533826424772	533810	0	18
25	17270	107025	17270	1080424772	533810424837	533828	0	19
25	17270	107025	179999999990424837		533828424785	533902	0	20
25	17270	108025	17270	1090424772	533810424816	533716	0	21
25	17270	108025	17270	1240424816	533716424876	533770	0	22
25	17270	108025	179999999990424876		533770424837	533828	0	23
25	17270	109025	17270	1110424690	533779424756	533719	0	24
25	17270	109025	17270	1110424756	533719424816	533716	0	25
25	17270	111025	17270	1170424646	533712424708	533655	0	26
25	17270	111025	17270	1190424708	533655424747	533672	0	27
25	17270	111025	17270	1180424747	533672424819	533704	0	28
25	17270	111025	17270	1240424819	533704424816	533716	0	29
25	17270	117025	179999999990424608		533734424576	533670	0	30
25	17270	117025	179999999990424576		533670424656	533655	0	31
25	17270	117025	179999999990424656		533655424693	533577	0	32
25	17270	117025	17270	1300424693	533577424713	533601	0	33
25	17270	117025	17270	1190424713	533601424687	533643	0	34
25	17270	117025	17270	1190424687	533643424708	533655	0	35
25	17270	118025	17270	1190424747	533672424756	533648	0	36
25	17270	118025	17270	1200424756	533648424771	533644	0	37
25	17270	118025	17270	1210424771	533644424783	533653	0	38
25	17270	118025	17270	1220424783	533653424818	533678	0	39
25	17270	118025	17270	1240424818	533678424819	533704	0	40
25	17270	119025	17270	1300424713	533601424735	533600	0	41
25	17270	119025	17270	1200424735	533600424756	533648	0	42
25	17270	120025	17270	1300424735	533600424772	533609	0	43
25	17270	120025	17270	1310424772	533609424782	533619	0	44
25	17270	120025	17270	1210424782	533619424771	533644	0	45

TABLE 3. 1964-1969 USER DEMAND AREAS BY APPROXIMATE GEOCODE IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	121025	17270	1310424782	533619424809	533621	0	46
25	17270	121025	17270	1220424809	533621424783	533653	0	47
25	17270	122025	17270	1230424809	533621424823	533665	0	48
25	17270	122025	17270	1240424823	533665424818	533678	0	49
25	17270	123025	17270	1310424809	533621424840	533592	0	50
25	17270	123025	17270	1400424840	533592424840	533616	0	51
25	17270	123025	17270	1240424840	533616424823	533665	0	52
25	17270	124025	17270	1400424840	533616424869	533615	0	53
25	17270	124025	17270	1400424869	533615424882	533639	0	54
25	17270	124025	17270	1250424882	533639424874	533661	0	55
25	17270	124025	17270	1260424874	533661424895	533764	0	56
25	17270	124025	179999999990424895		533764424876	533770	0	57
25	17270	125025	17270	1460424882	533639424918	533642	0	58
25	17270	125025	17270	1260424918	533642424907	533666	0	59
25	17270	125025	17270	1260424907	533666424874	533661	0	60
25	17270	126025	17270	1470424918	533642424934	533644	0	61
25	17270	126025	17270	1480424934	533644424960	533650	0	62
25	17270	126025	17270	1620424960	533650424947	533726	0	63
25	17270	126025	17270	1620424947	533726424968	533726	0	64
25	17270	126025	17270	1680424968	533726424963	533764	0	65
25	17270	126025	179999999990424963		533764424895	533764	0	66
25	17270	130025	179999999990424693		533577424721	533519	0	67
25	17270	130025	17270	1340424721	533519424797	533547	0	68
25	17270	130025	17270	1320424797	533547424784	533577	0	69
25	17270	130025	17270	1310424784	533577424772	533609	0	70
25	17270	131025	17270	1320424784	533577424840	533592	0	71
25	17270	132025	17270	1330424797	533547424838	533524	0	72
25	17270	132025	17270	1400424838	533524424840	533592	0	73
25	17270	133025	17270	1340424797	533547424805	533496	0	162
25	17270	133025	17270	1340424805	533496424850	533494	0	74
25	17270	133025	17270	1410424850	533494424838	533524	0	75
25	17270	134025	179999999990424721		533519424736	533488	0	76
25	17270	134025	17270	1380424736	533488424740	533497	0	77
25	17270	134025	17270	1380424740	533497424779	533468	0	78
25	17270	134025	17270	1350424779	533468424828	533457	0	79
25	17270	134025	17270	1390424828	533457424850	533494	0	80
25	17270	135025	17270	1380424779	533468424770	533441	0	81
25	17270	135025	17270	1360424770	533441424819	533431	0	82
25	17270	135025	17270	1390424819	533431424828	533457	0	83
25	17270	136025	17270	1380424770	533441424751	533344	0	84
25	17270	136025	179999999990424751		533344424819	533364	0	85
25	17270	136025	17270	1390424819	533364424819	533431	0	86
25	17270	138025	179999999990424736		533488424757	533448	0	87
25	17270	138025	179999999990424757		533448424751	533344	0	88
25	17270	139025	179999999990424819		533364424944	533400	0	89

TABLE 3. 1964-1969 USER DEMAND AREAS BY APPROXIMATE GEOCODE IN DIME
FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101
AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT
ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	139025	17270	1440424944	533400424884	533447	0	90
25	17270	139025	17270	1420424884	533447424850	533494	0	91
25	17270	140025	17270	1410424838	533524424916	533570	0	92
25	17270	140025	17270	1470424916	533570424907	533585	0	93
25	17270	140025	17270	1460424907	533585424869	533615	0	94
25	17270	141025	17270	1420424850	533494424930	533537	0	95
25	17270	141025	17270	1480424930	533537424916	533570	0	96
25	17270	142025	17270	1440424884	533447424930	533537	0	97
25	17270	144025	179999999990424944		533400424997	533422	0	98
25	17270	144025	17270	1510424997	533422424952	533498	0	99
25	17270	144025	17270	1500424952	533498424930	533537	0	100
25	17270	146025	17270	1470424907	533585424918	533642	0	101
25	17270	147025	17270	1480424916	533570424932	533596	0	102
25	17270	147025	17270	1480424932	533596424934	533644	0	103
25	17270	148025	17270	1500424930	533537424967	533571	0	104
25	17270	148025	17270	1490424967	533571424972	533609	0	105
25	17270	148025	17270	1520424972	533609424980	533645	0	106
25	17270	148025	17270	1620424980	533645424960	533650	0	107
25	17270	149025	17270	1500424967	533571425003	533570	0	108
25	17270	149025	17270	1540425003	533570425000	533595	0	109
25	17270	149025	17270	1520425000	533595424972	533609	0	110
25	17270	150025	17270	1510424952	533498425011	533529	0	111
25	17270	150025	17270	1540425011	533529425003	533570	0	112
25	17270	151025	179999999990424997		533422425025	533434	0	113
25	17270	151025	17270	1580425025	533434425018	533484	0	114
25	17270	151025	17270	1580425018	533484425011	533529	0	115
25	17270	152025	17270	1540425000	533595425011	533597	0	116
25	17270	152025	17270	1530425011	533597425038	533657	0	117
25	17270	152025	17270	1620425038	533657424980	533645	0	118
25	17270	153025	17270	1540425011	533597425033	533585	0	119
25	17270	153025	17270	1550425033	533585425070	533643	0	120
25	17270	153025	17270	1660425070	533643425038	533657	0	121
25	17270	154025	17270	1580425011	533529425023	533536	0	122
25	17270	154025	17270	1580425023	533536425069	533549	0	127
25	17270	154025	17270	1580425069	533549425115	533574	0	128
25	17270	154025	17270	1550425115	533574425033	533585	0	124
25	17270	155025	17270	1610425115	533574425093	533643	0	125
25	17270	155025	17270	1660425093	533643425070	533643	0	126
25	17270	158025	179999999990425025		533434425089	533463	0	131
25	17270	158025	179999999990425089		533463425282	533526	0	136
25	17270	158025	179999999990425282		533526425263	533559	0	137
25	17270	158025	179999999990425263		533559425240	533599	0	134
25	17270	158025	17270	1610425240	533599425115	533574	0	135
25	17270	161025	179999999990425240		533599425214	533644	0	138
25	17270	161025	17270	1700425214	533644425133	533666	0	139

TABLE 3. 1964-1969 USER DEMAND AREAS BY APPROXIMATE GEOCODE IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	161025	17270	1660425133	533666425093	533643	0	140
25	17270	162025	17270	1660425038	533657425036	533695	0	141
25	17270	162025	17270	1640425036	533695425036	533707	0	142
25	17270	162025	17270	1630425036	533707425003	533714	0	143
25	17270	162025	17270	1680425003	533714424968	533726	0	144
25	17270	163025	17270	1640425036	533707425059	533746	0	145
25	17270	163025	17270	1680425059	533746425013	533741	0	146
25	17270	163025	17270	1680425013	533741425002	533714	0	147
25	17270	164025	17270	1660425036	533695425071	533702	0	148
25	17270	164025	17270	1650425071	533702425067	533731	0	149
25	17270	164025	17270	1670425067	533731425059	533746	0	150
25	17270	165025	17270	1660425071	533702425113	533698	0	151
25	17270	165025	17270	1700425113	533698425109	533737	0	152
25	17270	165025	17270	1670425109	533737425067	533731	0	153
25	17270	166025	17270	1700425133	533666425113	533698	0	154
25	17270	167025	17270	1690425109	533737425077	533774	0	155
25	17270	167025	17270	1680425077	533774425059	533746	0	156
25	17270	168025	179999999990425077		533774424963	533764	0	157
25	17270	169025	17270	1700425109	533737425151	533752	0	158
25	17270	169025	179999999990425151		533752425143	533765	0	159
25	17270	169025	179999999990425143		533765425077	533774	0	160
25	17270	170025	179999999990425214		533644425151	533752	0	161

TABLE 4. 1970-1979 USER DEMAND AREAS BY APPROXIMATE GEOCODE IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	101025	17270	1030424535	533846424612	533882	0	01
25	17270	101025	17270	1040424612	533882424727	533953	0	02
25	17270	101025	179999999990424727		533953424644	534026	0	03
25	17270	101025	179999999990424644		534026424629	534039	0	04
25	17270	101025	179999999990424629		534039424500	533899	0	05
25	17270	101025	179999999990424500		533899424535	533846	0	06
25	17270	103025	179999999990424535		533846424580	533777	0	07
25	17270	103025	17270	1060424580	533777424635	533795	0	08
25	17270	103025	17270	1040424635	533795424612	533882	0	09
25	17270	104025	17270	1060424635	533795424750	533855	0	10
25	17270	104025	17270	1070424750	533855424785	533902	0	11
25	17270	104025	179999999990424785		533902424727	533953	0	12
25	17270	106025	179999999990424580		533777424608	533734	0	13
25	17270	106025	17270	1170424608	533734424646	533712	0	14
25	17270	106025	17270	1110424646	533712424690	533779	0	15
25	17270	106025	17270	1090424690	533779424757	533826	0	16
25	17270	106025	17270	1070424757	533826424750	533855	0	17
25	17270	107025	17270	1090424757	533826424772	533810	0	18
25	17270	107025	17270	1080424772	533810424837	533828	0	19
25	17270	107025	179999999990424837		533828424785	533902	0	20
25	17270	108025	17270	1090424772	533810424816	533716	0	21
25	17270	108025	17270	1240424816	533716424876	533770	0	22
25	17270	108025	179999999990424876		533770424837	533828	0	23
25	17270	109025	17270	1110424690	533779424756	533719	0	24
25	17270	109025	17270	1110424756	533719424816	533716	0	25
25	17270	111025	17270	1170424646	533712424708	533655	0	26
25	17270	111025	17270	1190424708	533655424747	533672	0	27
25	17270	111025	17270	1180424747	533672424819	533704	0	28
25	17270	111025	17270	1240424819	533704424816	533716	0	29
25	17270	117025	179999999990424608		533734424576	533670	0	30
25	17270	117025	179999999990424576		533670424656	533655	0	31
25	17270	117025	179999999990424656		533655424693	533577	0	32
25	17270	117025	17270	1300424693	533577424713	533601	0	33
25	17270	117025	17270	1190424713	533601424687	533643	0	34
25	17270	117025	17270	1190424687	533643424708	533655	0	35
25	17270	118025	17270	1190424747	533672424756	533648	0	36
25	17270	118025	17270	1200424756	533648424771	533644	0	37
25	17270	118025	17270	1210424771	533644424783	533653	0	38
25	17270	118025	17270	1220424783	533653424818	533678	0	39
25	17270	118025	17270	1240424818	533678424819	533704	0	40
25	17270	119025	17270	1300424713	533601424735	533600	0	41
25	17270	119025	17270	1200424735	533600424756	533648	0	42
25	17270	120025	17270	1300424735	533600424772	533609	0	43
25	17270	120025	17270	1310424772	533609424782	533619	0	44
25	17270	120025	17270	1210424782	533619424771	533644	0	45

TABLE 4. 1970-1979 USER DEMAND AREAS BY APPROXIMATE GEOCODES IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	121025	17270	1310424782	533619424809	533621	0	46
25	17270	121025	17270	1220424809	533621424783	533653	0	47
25	17270	122025	17270	1230424809	533621424823	533665	0	48
25	17270	122025	17270	1240424823	533665424818	533678	0	49
25	17270	123025	17270	1310424809	533621424840	533592	0	50
25	17270	123025	17270	1400424840	533592424840	533616	0	51
25	17270	123025	17270	1240424840	533616424823	533665	0	52
25	17270	124025	17270	1400424840	533616424869	533615	0	53
25	17270	124025	17270	1460424869	533615424882	533639	0	54
25	17270	124025	17270	1250424882	533639424874	533661	0	55
25	17270	124025	17270	1260424874	533661424895	533764	0	56
25	17270	124025	1799999999990424895		533764424876	533770	0	57
25	17270	125025	17270	1460424882	533639424918	533642	0	58
25	17270	125025	17270	1260424918	533642424907	533666	0	59
25	17270	125025	17270	1260424907	533666424874	533661	0	60
25	17270	126025	17270	1470424918	533642424934	533644	0	61
25	17270	126025	17270	1480424934	533644424960	533650	0	62
25	17270	126025	17270	1620424960	533650424947	533726	0	63
25	17270	126025	17270	1620424947	533726424968	533726	0	64
25	17270	126025	17270	1680424968	533726424963	533764	0	65
25	17270	126025	1799999999990424963		533764424895	533764	0	66
25	17270	130025	1799999999990424693		533577424721	533519	0	67
25	17270	130025	17270	1340424721	533519424797	533547	0	68
25	17270	130025	17270	1320424797	533547424784	533577	0	69
25	17270	130025	17270	1310424784	533577424772	533609	0	70
25	17270	131025	17270	1320424784	533577424840	533592	0	71
25	17270	132025	17270	1330424797	533547424838	533524	0	72
25	17270	132025	17270	1400424838	533524424840	533592	0	73
25	17270	133025	17270	1340424797	533547424805	533496	0	162
25	17270	133025	17270	1340424805	533496424850	533494	0	74
25	17270	133025	17270	1410424850	533494424838	533524	0	75
25	17270	134025	1799999999990424721		533519424736	533488	0	76
25	17270	134025	17270	1380424736	533488424740	533497	0	77
25	17270	134025	17270	1380424740	533497424779	533468	0	78
25	17270	134025	17270	1350424779	533468424828	533457	0	79
25	17270	134025	17270	1390424828	533457424850	533494	0	80
25	17270	135025	17270	1380424779	533468424770	533441	0	81
25	17270	135025	17270	1360424770	533441424819	533431	0	82
25	17270	135025	17270	1390424819	533431424828	533457	0	83
25	17270	136025	17270	1380424770	533441424751	533344	0	84
25	17270	136025	1799999999990424751		533344424819	533364	0	85
25	17270	136025	17270	1390424819	533364424819	533431	0	86
25	17270	138025	1799999999990424736		533488424757	533448	0	87
25	17270	138025	1799999999990424757		533448424751	533344	0	88
25	17270	139025	1799999999990424819		533364424944	533400	0	89

TABLE 4. 1970-1979 USER DEMAND AREAS BY APPROXIMATE GEOCODES IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	139025	17270	1440424944	533400424884	533447	0	90
25	17270	139025	17270	1420424884	533447424850	533494	0	91
25	17270	140025	17270	1410424838	533524424916	533570	0	92
25	17270	140025	17270	1470424916	533570424907	533585	0	93
25	17270	140025	17270	1460424907	533585424869	533615	0	94
25	17270	141025	17270	1420424850	533494424930	533537	0	95
25	17270	141025	17270	1480424930	533537424916	533570	0	96
25	17270	142025	17270	1440424884	533447424930	533537	0	97
25	17270	144025	179999999990424944		533400424997	533422	0	98
25	17270	144025	17270	1510424997	533422424952	533498	0	99
25	17270	144025	17270	1500424952	533498424930	533537	0	100
25	17270	146025	17270	1470424907	533585424918	533642	0	101
25	17270	147025	17270	1480424916	533570424932	533596	0	102
25	17270	147025	17270	1480424932	533596424934	533644	0	103
25	17270	148025	17270	1500424930	533537424967	533571	0	104
25	17270	148025	17270	1490424967	533571424972	533609	0	105
25	17270	148025	17270	1520424972	533609424980	533645	0	106
25	17270	148025	17270	1620424980	533645424960	533650	0	107
25	17270	149025	17270	1500424967	533571425003	533570	0	108
25	17270	149025	17270	1540425003	533570425000	533595	0	109
25	17270	149025	17270	1520425000	533595424972	533609	0	110
25	17270	150025	17270	1510424952	533498425011	533529	0	111
25	17270	150025	17270	1540425011	533529425003	533570	0	112
25	17270	151025	179999999990424997		533422425025	533434	0	113
25	17270	151025	17270	1580425025	533434425018	533484	0	114
25	17270	151025	17270	1570425018	533484425011	533529	0	115
25	17270	152025	17270	1540425000	533595425011	533597	0	116
25	17270	152025	17270	1530425011	533597425038	533657	0	117
25	17270	152025	17270	1620425038	533657424980	533645	0	118
25	17270	153025	17270	1540425011	533597425033	533585	0	119
25	17270	153025	17270	1550425033	533585425070	533643	0	120
25	17270	153025	17270	1660425070	533643425038	533657	0	121
25	17270	154025	17270	1570425011	533529425023	533536	0	122
25	17270	154025	17270	1560425023	533536425033	533585	0	123
25	17270	155025	17270	1560425033	533585425115	533574	0	124
25	17270	155025	17270	1610425115	533574425093	533643	0	125
25	17270	155025	17270	1660425093	533643425070	533643	0	126
25	17270	156025	17270	1570425023	533536425069	533549	0	127
25	17270	156025	17270	1570425069	533549425115	533574	0	128
25	17270	157025	17270	1580425018	533484425082	533484	0	129
25	17270	157025	17270	1580425082	533484425263	533559	0	133
25	17270	157025	179999999990425263		533559425240	533599	0	134
25	17270	157025	17270	1610425240	533599425115	533574	0	135
25	17270	158025	179999999990425025		533434425089	533463	0	131
25	17270	158025	179999999990425089		533463425282	533526	0	136

TABLE 4. 1970-1979 USER DEMAND AREAS BY APPROXIMATE GEOCODES IN DIME FORMAT: STATE,COUNTY,CITY,DEMAND AREA = 25, 17,270, 101 AND THE BOUNDARY BETWEEN AREAS GIVEN BY LINE SEGMENT ENDPOINTS, EG: 424535 533884 AND 424612 533882

25	17270	158025	179999999990425282	533526425263	533559	0	137
25	17270	161025	179999999990425240	533599425214	533644	0	138
25	17270	161025	17270 1700425214	533644425133	533666	0	139
25	17270	161025	17270 1660425133	533666425093	533643	0	140
25	17270	162025	17270 1660425038	533657425036	533695	0	141
25	17270	162025	17270 1640425036	533695425036	533707	0	142
25	17270	162025	17270 1630425036	533707425003	533714	0	143
25	17270	162025	17270 1680425003	533714424968	533726	0	144
25	17270	163025	17270 1640425036	533707425059	533746	0	145
25	17270	163025	17270 1680425059	533746425013	533741	0	146
25	17270	163025	17270 1680425013	533741425003	533714	0	147
25	17270	164025	17270 1660425036	533695425071	533702	0	148
25	17270	164025	17270 1650425071	533702425067	533731	0	149
25	17270	164025	17270 1670425067	533731425059	533746	0	150
25	17270	165025	17270 1660425071	533702425113	533698	0	151
25	17270	165025	17270 1700425113	533698425109	533737	0	152
25	17270	165025	17270 1670425109	533737425067	533731	0	153
25	17270	166025	17270 1700425133	533666425113	533698	0	154
25	17270	167025	17270 1690425109	533737425077	533774	0	155
25	17270	167025	17270 1680425077	533774425059	533746	0	156
25	17270	168025	179999999990425077	533774424963	533764	0	157
25	17270	169025	17270 1700425109	533737425151	533752	0	158
25	17270	169025	179999999990425151	533752425143	533765	0	159
25	17270	169025	179999999990425143	533765425077	533774	0	160
25	17270	170025	179999999990425214	533644425151	533752	0	161

TABLE 5. WELL USE AND PUMPING RATES, AND TOTAL USER DEMAND

Month and Year	Mo Avg GPM A2,B,C2	Mo Avg GPM E on	Other Wells on	Total User Demand	Fract G/H on	Fract E on	Fract E off
Oct.64	1018	681	D,F,G	2729	0.994	0.429	0.571
Nov.64	1590	701	F,G	2493	0.783	0.387	0.613
Dec.64	1528	715	F,G	2514	0.865	0.425	0.575
Dec.65	1493	625	G	2375	0.965	0.114	0.886
Jan.66	1493	646	G	2438	1.000	0.119	0.881
Feb.66	1618	597	G	2465	1.000	0.082	0.918
Mar.66	1806	0	G	2535	1.000	0.000	1.000
Apr.66	1449	0	D,F,G	2972	1.000	0.000	1.000
May 66	2250	0	G	2903	0.945	0.000	1.000
June66	1713	590	D,F,G	3389	1.000	0.090	0.910
July66	2116	806	D,F,G	4222	1.000	0.610	0.390
Aug.66	2944	715	G	3875	1.000	0.335	0.665
Sep.66	1827	660	F,G	3090	1.000	0.507	0.493
Oct.66	1625	674	F,G	2861	1.000	0.455	0.545
Nov.66	1889	681	F,G	2847	0.567	0.488	0.512
Dec.66	1819	681	G	2604	0.823	0.365	0.635
Jan.67	1625	729	F,G	2674	0.823	0.384	0.616
Feb.67	1653	715	F,G	2965	1.000	0.536	0.464
Mar.67	1708	674	F,G	2653	0.387	0.333	0.667
July67	2150	0	D,F,H	3160	0.097	0.000	1.000
Aug.67	1609	0	D,F,H	3486	0.258	0.000	1.000
		465	D,F,G,H		0.645	0.250	0.750
Sep.67	1282	465	D,F,G	3569	0.133	0.925	0.075
	1972	465	F,H		0.100	0.867	0.133
	1282	465	D,F,G,H		0.767	0.552	0.448
Oct.67	977	458	D,F,G,H	3431	1.000	0.710	0.290
Nov.67	1095	444	D,F,G,H	3361	0.833	0.920	0.080
Dec.67	1852	451	D,F,G,H	3333	0.139	1.000	0.000

Well D pumped approximately 690 GPM, when operating.
Well F pumped approximately 236 GPM, when operating.
Well G pumped approximately 800 GPM, when operating.
Well H pumped approximately 400 GPM, when operating.

Month and Year	Mo Avg GPM A2,B,C2	Mo Avg GPM E on	Other Wells on	Total User Demand	Fract G/H on	Fract E on	Fract E off
Apr.68	1734	486	D,F,G	3583	0.633	0.789	0.211
May 68	1567	458	D,F,G	3694	1.000	0.871	0.129
June68	1456	472	D,F,G	3646	1.000	0.997	0.003
July68	2109	479	D,F,G	4257	1.000	0.874	0.126
Aug.68	2220	472	D,F,G	4347	1.000	0.884	0.116
Sep.68	1866	444	D,F,G	3944	0.997	0.823	0.177
Oct.68	1602	438	D,F,G	3646	1.000	0.739	0.261
Nov.68	2160	438	F,G	3389	0.827	0.847	0.153
Dec.68	2340	472	G	3306	0.823	0.765	0.235
Jan.69	2306	479	F,G	3431	0.813	0.587	0.413
Feb.69	2181	479	F,G	3472	1.000	0.536	0.464
Mar.69	1449	479	D,F,G	3472	1.000	0.600	0.400
Apr.69	1470	479	D,F,G	3472	1.000	0.563	0.437
May 69	1909	493	D,F,G	3660	1.000	0.642	0.358
June69	2414	486	D,F,G	4507	1.000	0.750	0.250
July69	2241	514	D,F,G	4375	1.000	0.784	0.216
Aug.69	2046	500	D,F,G	4201	1.000	0.868	0.132
Sep.69	2354	493	F,G	3694	1.000	0.703	0.297
Oct.69	2653	458	F,G	3271	0.016	1.000	0.000

Well D pumped approximately 690 GPM, when operating.
Well F pumped approximately 236 GPM, when operating.
Well G pumped approximately 800 GPM, when operating.
Well H pumped approximately 400 GPM, when operating.

Month and Year	Mo Avg GPM A2,B,C2	Mo Avg GPM E on	Other Wells on	Total User Demand	Fract G/H on	Fract E on	Fract E off
Apr.70	1789	486	D,F,G	3347	0.500	0.707	0.293
May 70	1699	500	D,F,G	3583	0.703	0.794	0.206
June70	2352	493	D,F,G	4410	1.000	0.853	0.147
July70	2178	472	D,F,G	4229	0.984	0.721	0.279
Aug.70	2053	458	D,F,G	4139	1.000	0.787	0.213
Sep.70	1512	465	D,F,G	2910	1.000	0.567	0.433
Oct.70	1782	465	D,F,G	3236	0.500	0.800	0.200
Nov.70	2229	451	F,G	3042	0.327	0.745	0.255
Dec.70	1484	458	D,F,G	2965	0.[illegible]22	0.777	0.223
May 71	1859	472	D,F,G	3278	0.100	1.000	0.000
July71	2498	493	D,F,G	4431	0.735	0.877	0.123
Aug.71	2354	486	F,G	3840	1.000	0.945	0.055
Sep.71	2264	472	F,G	3528	0.930	0.570	0.430
Oct.71	1875	493	F,G	3153	0.823	0.769	0.231
Nov.71	1861	465	F,G	2924	0.550	0.970	0.030
Dec.71	1632	465	F,G	2917	0.813	1.000	0.000
Jan.72	1137	465	D,F,G	3132	0.832	0.996	0.004
Feb.72	1715	472	F,G	3028	1.000	0.576	0.424
Mar.72	1123	479	D,F,G	3097	0.913	0.611	0.389
Apr.72	1366	486	D,F,G	3167	0.540	1.000	0.000
Aug.74	2206	479	D,F,G	4396	0.865	1.000	0.000
			D,F,G,H		0.106	0.939	0.061
Sep.74	1833	472	F,G	3611	0.077	1.000	0.000
			F,H		0.010	1.000	0.000
			F,G,H		0.913	1.000	0.000
Oct.74	1715	507	F,G	3542	0.029	1.000	0.000
			F,H		0.010	1.000	0.000
			F,G,H		0.961	1.000	0.000
Nov.74	1695	438	F,G	3319	0.357	1.000	0.000
			F,H		0.010	1.000	0.000
			F,G,H		0.620	1.000	0.000
Dec.74	2000	444	F,G	3028	0.271	1.000	0.000
			F,G,H		0.132	1.000	0.000

Well D pumped approximately 690 GPM, when operating.
Well F pumped approximately 236 GPM, when operating.
Well G pumped approximately 800 GPM, when operating.
Well H pumped approximately 400 GPM, when operating.

Month and Year	Mo Avg GPM A2,B,C2	Mo Avg GPM E on	Other Wells on	Total User Demand	Fract G/H on	Fract E on	Fract E off
May 75	2303	444	D,G	3799	0.465	0.993	0.007
June75	1977	438	D,F,G	4090	0.923	1.000	0.000
			D,F,G,H		0.077	1.000	0.000
July75	1963	438	D,F,G	4410	0.042	1.000	0.000
			D,F,G,H		0.958	1.000	0.000
Aug.75	1706	444	D,F,G	4007	0.423	1.000	0.000
			D,F,G,H		0.577	1.000	0.000
Sep.75	1289	444	D,F,G	3556	0.690	1.000	0.000
			D,F,H		0.010	1.000	0.000
			D,F,G,H		0.300	1.000	0.000
Oct.75	1602	451	D,F,G	3424	0.626	1.000	0.000
Nov.75	1852	451	D,F,G	3528	0.417	1.000	0.000
Dec.75	1449	458	D,F,G	3521	0.961	1.000	0.000
Jan.76	1317	444	D,F,G	3417	1.000	1.000	0.000
Feb.76	1637	451	D,F,G	3722	0.966	1.000	0.000
Mar.76	1859	431	D,F,G	3611	0.555	0.994	0.006
June76	2713	444	D,F,G	4861	0.003	1.000	0.000
			D,F,H		0.027	1.000	0.000
			D,F,G,H		0.673	1.000	0.000
July76	2310	424	D,F,G,H	4215	1.000	1.000	0.000
Aug.76	2220	403	D,F,H	3944	0.310	1.000	0.000
			D,F,G,H		0.690	0.972	0.028
Sep.76	2220	403	D,F,H	3826	1.000	0.997	0.003
Oct.76	2116	396	D,F,H	3826	0.968	1.000	0.000
Jan.77	2067	403	D,F,G	4118	0.306	1.000	0.000
Feb.77	1928	389	D,F,G	3958	0.971	1.000	0.000
Mar.77	1887	389	D,F,G	3910	0.897	1.000	0.000
Apr.77	1866	389	D,F,G	3674	0.963	0.471	0.529
May 77	1574	639	D,F,G	3951	0.632	0.847	0.153
			D,F,G,H		0.368	1.000	0.000
June77	1755	639	D,F,G	3896	0.923	0.975	0.025
			D,F,G,H		0.077	1.000	0.000
July77	1928	632	D,F,G	4167	0.994	0.990	0.010
Aug.77	1748	618	D,F,G	3944	0.997	0.964	0.036
Sep.77	1706	590	D,F,G	3896	1.000	0.993	0.007
Oct.77	1602	597	D,F,G	3660	1.000	0.935	0.065
Nov.77	1581	604	D,F,G	3618	1.000	0.750	0.250
Dec.77	2081	0	D,F,G	3681	1.000	0.000	1.000

Well D pumped approximately 690 GPM, when operating.
Well F pumped approximately 236 GPM, when operating.
Well G pumped approximately 800 GPM, when operating.
Well H pumped approximately 400 GPM, when operating.

Month and Year	Mo Avg GPM A2,B,C2	Mo Avg GPM E on	Other Wells on	Total User Demand	Fract G/H on	Fract E on	Fract E off
Jan.78	2143	0	D,F,G	3750	1.000	0.000	1.000
Feb.78	2157	0	D,F,G	3771	1.000	0.000	1.000
Mar.78	2234	0	D,F,G	3806	0.181	0.000	1.000
			D,F,H		0.587	0.000	1.000
			D,F,G,H		0.232	0.000	1.000
Apr.78	2032	0	D,F,H	3944	0.200	0.000	1.000
			D,F,G,H		0.800	0.000	1.000
May 78	2067	688	D,F,G,H	4160	1.000	0.013	0.987
June78	2060	611	D,F,G,H	4729	0.993	1.000	0.000
July	2164	625	D,F,G,H	4847	1.000	1.000	0.000
Aug.78	2074	563	D,F,G,H	4292	1.000	0.287	0.713
Sep.78	2150	0	D,F,G	4194	0.020	0.000	1.000
			D,F,G,H		0.980	0.000	1.000
Oct.78	1824	0	D,F,G	3875	0.010	0.000	1.000
			D,F,G,H		0.990	0.000	1.000
Nov.78	1838	0	D,F,G	3889	0.003	0.000	1.000
			D,F,G,H		0.997	0.000	1.000
Dec.78	2067	535	D,F,G,H	4125	1.000	0.655	0.345
Jan.79	1935	0	D,F,H	4125	0.003	0.000	1.000
		556	D,F,G,H		0.997	0.311	0.689
Feb.79	2199	549	D,F,G,H	4333	1.000	0.054	0.946
Mar.79	1893	569	D,F,G,H	4486	1.000	1.000	0.000
Apr.79	1664	549	D,F,G,H	4201	1.000	0.967	0.033
May 79	1852	458	D,F,G,H	4181	0.700	1.000	0.000

Well D pumped approximately 690 GPM, when operating.
Well F pumped approximately 236 GPM, when operating.
Well G pumped approximately 800 GPM, when operating.
Well H pumped approximately 400 GPM, when operating.

TABLE 6. INPUT VARIABLES FOR THE COMPUTER MODELS

Variable	Symbol	Typical Value	Data Error Level
Pipe length	L	4200 ft.	50 ft.
Pipe diameter	D	12 in.	0.1 in.
Pipe connections	none	node number	none
Pipe roughness	C	80	40
Node water demand	Q_d	181 gal./min.	60 gal./min.
Node elevation	Z	161 ft.	1 ft.
Reservoir level	Z	312 ft.	1 ft.
Pump flow rate	Q_p	600 gal./min.	6 gal./min.
Pump total head	H_p	264 ft.	3 ft.
Well water level	Z	40 ft.	2 ft.
Check valve sites	none	pipe number	none
Pollutant Conc.	P	0.58 ppm	0.01 ppm

TABLE 7. ROUGHNESS COEFFICIENT SENSITIVITY ANALYSIS. RANDOM AND SYSTEMATIC CHANGES IN "C" AND THEIR AFFECTS ON THE EXPOSURE INDEX FOR OCTOBER, 1974.

Pipe No.	Orig. "C"	Rand. Diff. No.1	Rand. Diff. No.2	*	Node No.	Exp. Index	Rand. Diff. No.1	Rand. Diff. No.2	Sys. Diff. 1.25	Sys. Diff. 0.75
1	40	+18	-18	*	1	0.00	.00	.00	.00	.00
2	40	-17	+13	*	2	0.00	.00	.00	.00	.00
3	90	+ 5	-13	*	3	0.00	.00	.00	.00	.00
4	90	+15	+ 9	*	4	0.00	.00	.00	.00	.00
5	90	- 6	+17	*	5	0.00	.00	.00	.00	.00
6	90	+10	+ 5	*	6	0.00	.00	.00	.00	.00
7	80	+ 6	+13	*	7	0.00	.00	.00	.00	.00
8	80	+14	-17	*	8	0.00	.00	.00	.00	.00
9	40	+13	- 5	*	9	0.00	.00	.00	.00	.00
10	80	+ 3	- 2	*	10	0.00	.00	.00	.00	.00
11	50	+18	+11	*	11	0.00	.00	.00	.00	.00
12	40	-16	- 4	*	12	0.00	.00	.00	.00	.00
13	90	+16	+10	*	13	0.00	.00	.00	.00	.00
14	90	+ 8	- 9	*	14	0.00	.00	.00	.00	.00
15	90	+ 5	- 3	*	15	0.00	.00	.00	.00	.00
16	90	0	+ 7	*	16	0.00	.00	.00	.00	.00
17	90	+ 7	+10	*	17	0.00	.00	.00	.00	.00
18	80	+16	-16	*	18	0.00	.00	.00	.00	.00
19	40	-18	+18	*	19	0.00	.00	.00	.00	.00
20	40	+ 9	+13	*	20	0.00	.00	.00	.00	.00
21	40	- 3	+ 7	*	21	0.00	.00	.00	.00	.00
22	40	+ 9	-14	*	22	0.00	.00	.00	.00	.00
23	40	-14	+ 5	*	23	0.06	-.02	-.04	-.02	+.03
24	40	- 6	- 7	*	24	0.03	-.02	-.03	-.01	+.01
25	40	- 8	+12	*	25	0.03	-.02	-.03	-.01	+.01
26	40	+ 5	-16	*	26	0.15	.00	+.01	+.01	-.01
27	40	+ 8	+12	*	27	0.00	.00	.00	.00	.00
28	80	- 3	+16	*	28	0.02	.00	+.01	.00	-.01
29	80	-14	+ 9	*	29	0.02	.00	+.01	.00	-.01
30	80	- 8	-13	*	30	0.00	.00	.00	.00	.00
31	80	+ 4	0	*	31	0.00	.00	.00	.00	.00
32	90	-18	- 3	*	32	0.16	.00	-.05	-.03	+.02
33	40	-14	+ 7	*	33	0.44	+.02	-.04	-.04	+.02
34	90	- 6	- 3	*	34	0.45	+.27	+.02	-.07	+.06
35	90	- 8	-17	*	35	0.99	.00	.00	.00	.00
36	90	+15	+15	*	36	0.99	.00	.00	.00	.00
37	120	- 6	+17	*	38	0.99	.00	.00	.00	.00
38	40	- 1	+10	*	39	1.00	.00	.00	.00	.00
39	50	-16	- 2	*	40	0.87	-.04	-.01	-.01	.00
40	120	+14	+ 9	*	41	0.99	.00	.00	.00	.00
41	60	+12	+15	*	42	1.00	.00	.00	.00	.00
42	60	+ 8	+17	*	43	1.00	.00	.00	.00	.00
43	60	- 3	+ 2	*	44	1.00	.00	.00	.00	.00
44	60	-13	+12	*	45	1.00	.00	.00	.00	.00
45	50	- 4	- 5	*	46	0.61	+.04	+.03	.00	-.02
46	60	-12	+ 5	*	47	0.64	.00	.00	.00	-.05

Pipe No.	Orig. "C"	Rand. Diff. No.1	Rand. Diff. No.2	*
47	60	+15	+11	*
49	40	+14	+18	*
50	40	- 4	-13	*
51	50	+12	- 9	*
52	120	+17	+ 2	*
53	120	+16	-12	*
54	80	+ 4	- 6	*
55	120	+ 3	+ 1	*
56	80	- 2	+ 7	*
57	80	- 5	+ 1	*
58	110	0	+18	*
59	50	- 1	-14	*
60	100	- 6	+ 1	*
61	100	+17	- 8	*
62	100	- 5	+18	*
63	50	+11	+10	*
64	80	+14	+15	*
65	90	-12	0	*
66	90	0	- 5	*
67	90	+15	+ 7	*
68	90	+14	+17	*
69	90	- 4	- 6	*
70	110	+ 9	+ 7	*
71	90	+ 9	-18	*
72	100	-16	+ 7	*
73	110	-13	+ 2	*
74	110	-12	+ 6	*
75	50	- 8	-16	*
76	40	+12	+ 4	*
77	110	+14	+14	*
78	110	- 7	+ 8	*
79	110	+10	+ 3	*
80	120	-11	+ 9	*
81	110	+10	+ 1	*
82	110	+13	+ 6	*
86	110	- 5	+12	*
87	80	+ 4	+ 6	*
88	100	+15	0	*
89	80	+16	-14	*
90	120	- 9	-16	*
91	120	0	+10	*
92	90	+ 8	- 9	*
93	90	+ 2	- 6	*
94	90	-16	0	*
95	120	- 1	-12	*
96	80	-10	+ 5	*
97	80	-15	- 5	*
98	120	+12	0	*
99	120	- 5	- 8	*
100	120	+ 1	+14	*
101	120	-12	+11	*
102	120	+14	+ 1	*

Node No.	Exp. Index	Rand. Diff. No.1	Rand. Diff. No.2	Sys. Diff. 1.25	Sys. Diff. 0.75
48	0.90	.00	.00	+.02	-.02
49	0.99	.00	.00	.00	.00
50	0.99	.00	.00	.00	.00
51	1.00	.00	.00	.00	.00
52	0.90	.00	.00	+.02	-.02
53	0.90	.00	.00	+.02	-.02
54	0.98	.00	.00	.00	.00
55	0.91	.00	+.01	+.02	-.02
56	0.99	.00	.00	.00	.00
57	1.00	.00	.00	.00	.00
58	1.00	.00	.00	.00	.00
61	0.91	.00	+.01	+.02	-.02
62	0.22	.00	.00	+.01	-.01
63	0.22	.00	.00	+.01	-.01
64	0.30	-.02	+.02	+.01	-.01
65	0.30	-.02	+.02	+.01	-.01
66	0.91	.00	.00	+.02	-.02
67	0.30	-.02	+.02	+.01	-.01
68	0.29	-.01	+.01	+.01	-.01
69	0.30	-.02	+.02	+.01	-.01
70	0.30	-.02	+.02	+.01	-.01
71	0.00	.00	.00	.00	.00
72	0.00	.00	.00	.00	.00

Exposure Index and Differences

N = 69					
AVG=	0.392	.002	.000	.000	-.002
S =	0.433	.034	.013	.013	.013

Pipe Roughness Coefficient

	Orig. "C"	Rand. Diff. No.1	Rand. Diff. No.2
N = 98			
Avg. =	81.3	1.5	2.1
S =	27.4	10.8	10.4

TABLE 8. 1980 WATER LEAKAGE SURVEY FINDINGS

Node No.	No. of Leaks	GPM (est./repaired)
1	14 service	140 / 30
3	4 service	40 / 20
6	3 main,8 ser.	140 / 60
7	3 service	30 / 20
8	1 service	10 / 10
9	3 service	30 / 20
11	1 misc,1 ser.	15 / 5
17	1 hydrant	3 / 0
19	1 main,7 ser.	90 / 30
20	1 hydrant	3 / 0
22	1 service	10 / 0
23	2 service	20 / 10
24	1 service	10 / 0
26	1 main,3 ser,1 hyd.	53 / 20
30	2 main,2 ser.	60 / 30
36	1 hydrant	3 / 3
39	1 main,1 hyd,1 ser,1 misc.	63 / 60
40	1 service	10 / 0
41	1 service	10 / 0
42	1 service	10 / 10
44	1 hydrant	3 / 0
46	1 service	10 / 10
48	1 service	10 / 0
57	1 hydrant	3 / 3
62	1 service	10 / 10
63	3 service	30 / 30
65	1 service	10 / 0
66	2 service	20 / 10
68	1 service	10 / 10
	Totals.....	866 / 401

TABLE 9. PEAKING FACTOR ANALYSIS.

Week: 6/21/70-6/27/70.

	Four Hour Pattern (8 AM/PM, peak/valley)							Eight Hour	
Days	-Mid	4 AM	8 AM	Noon	4 PM	8 PM	+Mid	Max.	Min.
Sun	0.91	0.81	0.95	1.29	1.14	0.99	0.82	1.22	0.85
Mon	0.76	0.74	1.03	1.18	1.12	1.13	0.82	1.15	0.74
Tues	0.80	0.73	1.01	1.28	1.14	1.12	0.77	1.21	0.77
Wed	0.77	0.69	1.03	1.26	1.20	1.09	0.73	1.24	0.73
Thur	0.74	0.72	1.00	1.18	1.17	1.17	0.78	1.18	0.73
Fri	0.87	0.84	1.14	1.19	1.01	1.00	0.88	1.19	0.86
Sat	X	X	X	X	X	X	X	X	X
N =	6	6	6	6	6	6	6	6	6
Avg =	0.81	0.76	1.03	1.23	1.13	1.08	0.80	1.20	0.78
s =	0.07	0.06	0.06	0.05	0.07	0.07	0.05	0.03	0.06

Week: 12/15/74-12/21/74.

	Four Hour Pattern(6:30 AM/PM, peak/vally)						Eight Hour	
Days	3 AM	6.5 AM	10 AM	2 PM	6.5 PM	11 PM	Max.	Min.
Sun	0.67	0.65	1.39	1.19	1.10	0.85	1.29	0.77
Mon	0.61	1.03	1.37	1.09	1.07	0.79	1.23	0.71
Tues	0.65	0.95	1.35	1.09	1.04	0.82	1.22	0.73
Wed	0.65	1.07	1.08	1.07	1.08	0.84	1.18	0.76
Thur	0.71	1.02	1.23	1.11	1.13	0.85	1.17	0.76
Fri	X	X	X	X	X	X	X	X
Sat	0.70	0.87	1.27	1.12	1.10	0.90	1.20	0.78
N =	6	6	6	6	6	6	6	6
Avg =	0.66	0.93	1.28	1.11	1.09	0.84	1.22	0.75
s =	0.04	0.16	0.12	0.04	0.03	0.04	0.04	0.03

Week: 10/30/83-11/5/83.

	Four Hour Pattern (6 AM/PM, peak/valley)							Eight Hour	
Days	-11PM	3 AM	6 AM	9 AM	1 PM	6 PM	11PM	Max.	Min.
Sun	X	X	X	1.11	1.14	0.98	0.79	1.12	X
Mon	0.79	0.77	1.00	1.41	1.12	X	X	1.27	0.78
Tues	X	X	X	X	1.17	1.08	0.82	X	X
Wed	0.82	0.71	0.99	1.19	1.06	1.04	0.77	1.13	0.76
Thur	0.77	0.76	0.94	1.16	1.00	0.96	1.03	1.11	0.77
Fri	1.03	1.03	1.03	1.13	1.09	1.01	0.84	1.00	1.00
Sat	0.84	0.87	1.06	1.14	1.19	1.06	X	1.16	0.87
N =	5	5	5	6	7	6	5	6	5
Avg =	0.85	0.83	1.00	1.19	1.11	1.02	0.85	1.13	0.84
s =	0.10	0.13	0.05	0.11	0.07	0.05	0.10	0.09	0.10

TABLE 10. GREAT OAKS FLUORIDE VALIDATION RESULTS

TEST NO.	NODE NO.	RAW DATA PPM	BASE LEVEL PPM	OBSERVED INJECTION CONC.-PPM	COMPUTER PREDICTION CONC.-PPM	SCALED PREDICTION CONC.-PPM	PREDICTION MINUS OBSERVED
101	136	1.55	.19	1.36	2.00	1.51	.15
102	136	1.55	.19	1.36	2.00	1.51	.15
103	108	0.85	.19	0.66	1.08	0.82	.16
104	134	1.48	.19	1.29	1.58	1.19	-.10
105	99	1.46	.19	1.27	1.40	1.05	-.22
106	114	0.19	.19	0.00	0.	0.	.06
107	106	0.18	.19	-0.01	1.08	0.82	.83*
108	118	0.18	.19	-0.01	0.	0.	.01
109	121	0.17	.19	-0.02	0.	0.	.02
110	112	0.18	.19	-0.01	0.	0.	.01
111	113	0.17	.19	-0.02	0.	0.	.02
112	110	0.21	.19	0.02	0.	0.	-.02
113	117	0.19	.19	0.00	0.	0.	.00
114	120	0.19	.19	0.00	0.	0.	.00
115	123	0.18	.19	-0,01	0.	0.	.01
116	109	0.18	.19	-0.01	1.08	0.82	.83
117	111	0.19	.19	0.00	0.	0.	.00
118	99	1.61	.19	1.42	1.40	1.05	-.37
119	105	1.59	.19	1.40	1.08	0.82	-.58*
120	104	0.26	.19	0.07	0.88	0.66	.59
121	98	0.20	.19	0.01	1.12	0.84	.83
122	91	1.15	.19	0.96	1.12	0.84	-.12
123	89	1.15	.19	0.96	1.12	0.84	-.12
124	88	1.41	.19	1.22	1.35	1.02	-.20
125	87	1.19	.19	1.00	1.16	0.88	-.12
126	101	0.19	.19	0.00	0.88	0.66	.66
127	102	0.19	.19	0.00	0.31	0.23	.23
128	103	0.80	.19	0.61	0.88	0.66	.05
129	100	0.66	.19	0.47	0.88	0.66	.19
130	101	0.21	.19	0.02	0.88	0.66	.64
131	97	0.85	.19	0.66	1.12	0.84	.18
132	96	0.68	.19	0.49	1.09	0.82	.33
133	95	0.41	.19	0.22	1.01	0.76	.54
134	93	0.79	.19	0.60	1.10	0.83	.23
135	92	1.07	.19	0.88	1.12	0.84	-.04
136	87	1.20	.19	1.01	1.16	0.88	-.13
137	86	1.16	.19	0.97	0.87	0.66	-.31
138	86	1.15	.19	0.96	0.87	0.66	-.30
139	85	0.52	.19	0.33	1.01	0.76	.43
140	73	0.38	.19	0.19	1.01	0.76	.57
141	84	1.32	.19	1.13	1.16	0.88	-.25
142	83	1.18	.19	0.99	0.28	0.21	-.78
143	68	1.29	.19	1.10	0.28	0.21	-.89
144	74	0.20	.19	0.01	0.	0.	-.01
145	77	1.21	.19	1.02	0.60	0.45	-.57
146	69	0.91	.19	0.72	0.61	0.46	-.26
147	114	0.21	.19	0.02	0.	0.	-.02
148	133	0.21	.21	0.00	0.	0.	.00
149	128	0.21	.21	0.00	0.	0.	.00
150	125	0.20	.21	-0.01	0.	0.	.01

* INVALID

TEST NO.	NODE NO.	RAW DATA PPM	BASE LEVEL PPM	OBSERVED INJECTION CONC.-PPM	COMPUTER PREDICTION CONC.-PPM	SCALED PREDICTION CONC.-PPM	PREDICTION MINUS OBSERVED
201	72	0.46	.19	0.27	1.01	0.76	.49
202	71	0.81	.19	0.62	0.84	0.64	.02
203	70	0.42	.19	0.23	0.86	0.65	.42
204	65	0.40	.19	0.21	0.86	0.65	.44
205	66	0.32	.19	0.13	0.86	0.65	.52
206	67	0.19	.19	0.00	0.85	0.64	.64*
207	64	0.20	.19	0.01	0.65	0.49	.48*
208	62	0.18	.19	-0.01	0.52	0.39	.40*
209	60	1.08	.19	0.89	0.52	0.39	-.50
210	61	0.88	.19	0.69	0.52	0.39	-.30
211	63	0.19	.19	0.00	0.52	0.39	.39*
212	57	1.03	.19	0.84	0.52	0.39	-.45
213	51	0.35	.19	0.16	0.12	0.09	-.07
214	47	0.55	.19	0.36	0.	0.	-.36
215	39	0.24	.21	0.03	0.	0.	-.03
216	42	0.23	.21	0.02	0.19	0.14	.12
217	45	0.20	.21	-0.01	0.23	0.17	.18
218	34	0.20	.21	-0.01	0.01	0.01	.02
219	32	0.21	.21	0.00	0.	0.	.00
220	15	0.23	.21	0.02	0.	0.	-.02
221	24	0.18	.21	-0.03	0.17	0.13	.16
222	28	0.19	.21	-0.02	0.01	0.01	.03
223	18	0.20	.21	-0.01	0.01	0.01	.02
224	21	0.18	.21	-0.03	0.17	0.13	.16
225	20	0.19	.21	-0.02	0.17	0.13	.15
226	12	0.20	.21	-0.01	0.	0.	.01
227	8	0.22	.21	0.01	0.	0.	-.01
228	9	0.22	.21	0.01	0.	0.	-.01
229	3	0.23	.21	0.02	0.	0.	-.02
230	16	0.23	.21	0.02	0.	0.	-.02
231	17	0.23	.21	0.02	0.01	0.01	-.02

N=74
AVERAGE=0.03
ROOT MEAN SQUARE=0.31

CHECK VALUES - BASE LEVELS

WELL	A	B	C
7	0.18	0.18	0.15
9	0.24	0.24	0.19
12	0.18	0.18	0.17
15	0.20	0.20	0.17
16	0.17	0.17	0.15

CHECK VALUES - TEST LEVELS

TEST NO.	A	B	C
112	0.21	0.20	0.16
117	0.19	0.19	0.14
121	0.20	0.20	0.15
131	0.85	0.85	0.63
133	0.41	0.42	0.27
143	1.29	1.30	0.93
212	1.03	1.04	0.72
214	0.55	0.55	0.35

PREDICTION MINUS OBSERVED
ERROR BANDS

ERROR RANGE	NUMBER IN RANGE	PERCENT
<±0.2	47	64
<±0.3	54	73
<±0.6	64	86

TABLE 11. WOBURN FLUORIDE VALIDATION RESULTS (3)

Site	Observed Concentration Lawrence (ppm)	UMASS (ppm)	Diff.	Second Predict. (ppm)	Diff.
Well E	0.058	0.06	0.00	0.07	-.01
2 Arlington	0.064	0.07	-.01	0.07	-.01
Booster Pump	0.059	0.07	-.01	0.07	-.01
Elm & West	0.468	0.48	-.01	0.08	0.41
Rag Rock Tank	0.138	0.14	0.00	0.07	0.07
14 Bedford	0.088	0.08	0.01	0.07	0.02
Mishawum & Garden	0.936	0.98	-.04	1.07	-.13
Main & Clinton	0.230	0.23	0.00	0.07	0.16
Cedar & Washington	0.976	1.05	-.07	1.07	-.09
14 Cedar	1.010	1.01	0.00	1.07	-.06
N Boston & N Indust	0.160	0.16	0.00	0.51	-.35
204 Salem	0.966	1.00	-.03	1.07	-.10
Booster Pump entry	0.080	0.07	0.01	0.07	0.01
66 Garfield	0.994	1.05	-.06	0.07	0.92
55 Bedford	0.148	0.15	0.00	0.07	0.08
369 Washington	0.610	0.66	-.05	1.07	-.46
44 Beach	0.551	0.53	0.02	0.51	0.04
51 School	0.810	0.80	0.01	0.18	0.63
130 Montvale	1.010	0.97	0.04	1.07	-.06
Washington&Montvale	1.42	1.50	-.08	1.07	0.35
89 Montvale	0.854	0.82	0.03	1.07	-.22
576 Main	0.195	0.18	0.02	0.07	0.13
Main&Middlesex Can	0.162	0.17	-.01	0.07	0.09
82 Beach	0.792	0.76	0.03	0.51	0.28
87 Olympia	0.170	0.16	0.01	1.07	-.90
2 Merrimac	0.554	0.58	-.03	0.51	0.04
15 Wyman	0.164	0.16	0.00	0.07	0.09
460 Wildwood	0.966	0.94	0.03	1.07	-.10
Well F	0.076	0.06	0.02	0.07	0.01
Forest Park	0.380	0.38	0.00	0.17	0.21
21 Clinton	0.200	0.21	-.01	0.07	0.13
12 Alfred	0.216	0.22	0.00	0.17	0.05
110 Pearl	0.194	0.19	0.00	0.08	0.11
9 Fowle	0.107	0.10	0.01	0.07	0.04
58 Water	0.066	0.07	0.00	0.07	0.00
22 Bow	0.978	1.10	-.12	1.07	-.09
10 Montvale	0.314	0.35	-.04	0.07	0.24
Garfield & Green	0.970	1.13	-.16	0.60	0.37
Montvale & Campbell	0.910	0.92	-.01	1.07	-.16
N Warren & Harrison	0.066	0.08	-.01	0.07	0.00
15 Fowle	0.954	1.04	-.09	0.07	0.88
Montvale & Green	0.974	1.02	-.05	1.07	-.10
Central & Utica	1.00	1.04	-.04	1.07	-.07
69 Wood	1.02	1.06	-.04	1.07	-.05
15 Auburn	1.00	1.10	-.10	1.07	-.07
108 Arlington	0.085	0.07	0.01	0.07	0.01

Site	Observed Concentration Lawrence (ppm)	UMASS (ppm)	Diff.	Second Predict. (ppm)	Diff.
Pleasant & Ellis	0.060	0.07	-.01	0.07	-.01
55 Salem	0.434	0.51	-.08	0.07	0.36
310 Main	0.316	0.37	-.05	0.07	0.25
7 Green	0.962	1.06	-.10	0.07	0.89
Salem & Main	0.220	0.25	-.03	0.07	0.15
80 Lexington	0.060	0.06	0.00	0.07	-.01
5 Harrison	0.077	0.10	-.02	0.07	0.01
Main & Pleasant	0.080	0.08	0.00	0.07	0.01
117 Green	0.978	1.03	-.05	1.07	-.10
Montvale & Washing	1.03	1.05	-.02	1.07	-.04
Washington & Pine	0.954	1.10	-.15	1.07	-.12
			N=57		N=57
			AVG=-0.02		AVG=0.07
			s= 0.04		s=0.29

PREDICTION VS OBSERVATION

Linear Regression:

Y = A X + B

X = Lawrence lab
Y = Re-prediction

A = 0.876
B =-0.0014
r = 0.778

Error Bands:

Error Range	Number in Range	Percent
=+0.1	32	56
=+0.2	41	72
=+0.3	46	81
=+0.5	52	91

TABLE 12. MONTHLY AVERAGING PROCESS FOR MAY, 1979.

FRACTION GH ON = .700 FRACTION E ON = 1.000 FRACTION E OFF = 0.000

NODE	EXPINDX	AVGEOFF	MAXEOFF	MINEOFF	AVGEON	MAXEON	MINEON
1	0.000	0.000	0.000		0.000	0.000	
2	0.000	0.000	0.000		0.000	0.000	
3	0.000	0.000	0.000		0.000	0.000	
4	0.000	0.000	0.000		0.000	0.000	
5	0.000	0.000	0.000		0.000	0.000	
6	0.000	0.000	0.000	0.000	0.000	0.000	0.000
7	0.000	0.000	0.000	.002	0.000	0.000	0.000
8	0.000	0.000	0.000	.054	0.000	0.000	0.000
9	0.000	0.000	0.000	0.000	0.000	0.000	0.000
10	0.000	0.000	0.000	0.000	0.000	0.000	0.000
11	0.000	0.000	0.000	0.000	0.000	0.000	0.000
12	0.000	0.000	0.000	0.000	0.000	0.000	0.000
13	0.000	0.000	0.000	0.000	0.000	0.000	0.000
15	0.000	0.000	0.000	0.000	0.000	0.000	0.000
17	0.000	0.000	0.000	0.000	0.000	0.000	0.000
18	0.000	0.000	0.000	0.000	0.000	0.000	0.000
19	0.000	0.000	0.000	0.000	0.000	0.000	0.000
20	0.000	0.000	0.000	0.000	0.000	0.000	0.000
21	0.000	0.000	0.000	0.000	0.000	0.000	0.000
22	0.000	0.000	0.000	0.000	0.000	0.000	0.000
23	.005	0.000	0.000	.056	0.000	0.000	.021
24	.003	0.000	0.000	.056	0.000	0.000	.012
25	.003	0.000	0.000	.056	0.000	0.000	.012
26	.052	0.000	0.000	.184	0.000	0.000	.224
27	0.000	0.000	0.000	0.000	0.000	0.000	0.000
28	0.000	0.000	0.000	0.000	0.000	0.000	0.000
30	0.000	0.000	0.000	0.000	0.000	0.000	0.000
31	0.000	0.000	0.000	0.000	0.000	0.000	0.000
32	.087	.108	.055	.162	.124	.067	.180
33	.248	.314	.267	.431	.337	.284	.443
34	.175	.215	.001	.490	.253	0.000	.496
35	.687	1.000	.899	1.000	1.000	.945	1.000
36	.692	1.000	.937	1.000	1.000	.966	1.000
38	.690	1.000	.918	1.000	1.000	.955	1.000
39	.700	1.000	1.000	1.000	1.000	1.000	1.000
40	.495	.690	.371	1.000	.713	.407	1.000
41	.700	1.000	1.000	1.000	1.000	1.000	1.000
42	.700	1.000	1.000	1.000	1.000	1.000	1.000
43	.700	1.000	1.000	1.000	1.000	1.000	1.000
44	.700	1.000	1.000	1.000	1.000	1.000	1.000
46	.270	.350	.137	.882	.328	.128	.699
47	.232	0.000	0.000	1.000	0.000	0.000	.995
48	.540	.862	.523	1.000	.827	.486	1.000
49	.700	1.000	1.000	1.000	1.000	1.000	1.000
50	.700	1.000	1.000	1.000	1.000	1.000	1.000
51	.700	1.000	1.000	1.000	1.000	1.000	1.000
52	.540	.862	.523	1.000	.827	.486	1.000
53	.540	.862	.523	1.000	.827	.486	1.000
54	.676	.985	.925	1.000	.980	.917	1.000
55	.546	.880	.531	1.000	.849	.490	1.000
56	.696	.998	.988	1.000	.997	.987	1.000
57	.700	1.000	1.000	1.000	1.000	1.000	1.000
58	.700	1.000	1.000	1.000	1.000	1.000	1.000
61	.546	.880	.531	1.000	.849	.490	1.000
62	.097	.079	.045	.262	.076	.042	.300
63	.097	.079	.045	.262	.076	.042	.300
64	.148	.170	.099	.349	.163	.091	.383
65	.148	.170	.099	.349	.163	.091	.383
66	.542	.870	.527	1.000	.837	.488	1.000
67	.148	.170	.099	.349	.163	.091	.383
68	.137	.150	.087	.330	.144	.080	.364
69	.148	.170	.099	.349	.163	.091	.383
70	.148	.170	.099	.349	.163	.091	.383
71	0.000	0.000	0.000	0.000	0.000	0.000	0.000
72	0.000	0.000	0.000	0.000	0.000	0.000	0.000
14	0.000	0.000	0.000	0.000	0.000	0.000	0.000
45	.700	1.000	1.000	1.000	1.000	1.000	1.000
16	0.000	0.000	0.000	0.000	0.000	0.000	0.000
29	0.000	0.000	0.000	0.000	0.000	0.000	0.000

TABLE 13. 1964-1969 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	OCT. 1964	NOV. 1964	DEC. 1964	DEC. 1965
1	.00	.00	.00	.01
2	.00	.00	.00	.01
3	.00	.00	.00	.01
4	.00	.00	.00	.01
5	.00	.00	.00	.01
6	.00	.00	.00	.00
7	.01	.01	.01	.04
8	.02	.02	.02	.06
9	.00	.01	.01	.02
10	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00
23	.05	.07	.07	.10
24	.04	.05	.05	.10
25	.05	.09	.10	.21
26	.19	.21	.22	.29
27	0.00	0.00	0.00	0.00
28	.01	.01	.02	.02
29	.01	.01	.02	.02
30	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00
32	.13	.11	.13	.15
33	.36	.31	.34	.42
34	.18	.20	.22	.32
35	.96	.76	.87	.97
36	.97	.78	.87	.97
38	.97	.78	.87	.97
39	.99	.78	.87	.97
40	.89	.75	.82	.96
41	.99	.78	.87	.97
42	.99	.78	.87	.97
43	.99	.78	.87	.97
44	.99	.78	.87	.97
45	.99	.78	.87	.97
46	.77	.67	.72	.93
47	.99	.78	.86	.96
48	.99	.78	.87	.97
49	.99	.78	.87	.97
50	.99	.78	.87	.97
51	.99	.78	.87	.97
52	.76	.62	.68	.78
53	.85	.69	.76	.85
54	.99	.78	.87	.97
55	.85	.69	.76	.85
58	.99	.78	.87	.97
61	.77	.63	.70	.80
62	.19	.21	.22	.29
63	.19	.21	.22	.29
64	.19	.21	.22	.29
65	.19	.21	.22	.29
66	.54	.46	.51	.60
67	.19	.21	.22	.29
68	.19	.21	.22	.29
69	.19	.21	.22	.29
70	.19	.21	.22	.29
71	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00

TABLE 13. 1964-1969 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1966	FEB. 1966	MAR. 1966	APR. 1966	MAY 1966	JUNE 1966	JULY 1966	AUG. 1966	SEP. 1966	OCT. 1966	NOV. 1966	DEC. 1966
1	.01	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
2	.01	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
3	.01	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
4	.01	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
5	.01	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
6	.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00
7	.03	.03	.03	0.00	.00	0.00	0.00	0.00	0.00	.00	.00	.01
8	.06	.06	.05	.02	.03	.01	0.00	0.00	.01	.02	.01	.03
9	.02	.02	.01	0.00	0.00	0.00	0.00	0.00	0.00	.00	0.00	.01
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	.10	.09	.08	.03	.04	.01	0.00	0.00	.02	.04	.02	.06
24	.09	.09	.08	.03	.04	.01	0.00	0.00	.02	.03	.02	.05
25	.19	.19	.19	.04	.07	.01	0.00	0.00	.03	.05	.03	.09
26	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.02	.01	.01	0.00	0.00	0.00	0.00	0.00	.00	.01	.00	.01
29	.02	.01	.01	0.00	0.00	0.00	0.00	0.00	.00	.01	.00	.01
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.16	.15	.13	.11	.11	.09	.04	.06	.12	.13	.07	.12
33	.42	.41	.38	.32	.31	.29	.24	.26	.32	.35	.19	.31
34	.30	.28	.23	.10	.11	.06	.03	.02	.09	.15	.08	.18
35	1.00	1.00	1.00	.91	.87	.81	.66	.70	.91	.95	.54	.81
36	1.00	1.00	1.00	.94	.90	.87	.76	.78	.94	.97	.55	.82
38	1.00	1.00	1.00	.93	.88	.84	.70	.74	.92	.96	.54	.81
39	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
40	.99	.98	.96	.82	.82	.64	.26	.42	.75	.85	.49	.77
41	1.00	1.00	1.00	1.00	.95	1.00	.97	1.00	1.00	1.00	.57	.82
42	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
43	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
44	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
45	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
46	.95	.95	.95	.68	.70	.48	.16	.27	.55	.67	.39	.69
47	1.00	1.00	1.00	.67	.68	.55	.27	.33	.56	.74	.45	.82
48	1.00	1.00	1.00	.99	.95	.84	.45	.59	.93	1.00	.57	.82
49	1.00	1.00	1.00	1.00	.95	1.00	.86	1.00	1.00	1.00	.57	.82
50	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
51	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
52	.80	.79	.79	.74	.71	.61	.32	.42	.69	.76	.43	.64
53	.88	.88	.87	.84	.80	.75	.54	.63	.81	.85	.48	.71
54	1.00	1.00	1.00	1.00	.95	1.00	.92	1.00	1.00	1.00	.57	.82
55	.88	.88	.87	.84	.80	.75	.54	.63	.81	.85	.48	.71
58	1.00	1.00	1.00	1.00	.95	1.00	1.00	1.00	1.00	1.00	.57	.82
61	.82	.81	.80	.75	.72	.65	.45	.54	.71	.76	.43	.65
62	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
63	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
64	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
65	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
66	.60	.60	.59	.51	.49	.42	.27	.33	.47	.52	.30	.47
67	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
68	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
69	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
70	.29	.28	.26	.15	.16	.06	.00	.02	.11	.16	.09	.19
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 13. 1964-1969 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1967	FEB. 1967	MAR. 1967	JULY 1967	AUG. 1967	SEP. 1967	OCT. 1967	NOV. 1967	DEC. 1967
1	.00	0.00	.00	0.00	.00	.00	.00	.00	0.00
2	.00	0.00	.00	0.00	.00	.00	.00	.00	0.00
3	.00	0.00	.00	0.00	.00	.00	.00	.00	0.00
4	.00	0.00	.00	0.00	.00	.00	.00	.00	0.00
5	.00	0.00	.00	0.00	.00	.00	.00	.00	0.00
6	.00	0.00	0.00	0.00	.00	.00	.00	.00	0.00
7	.01	.00	.00	0.00	.02	.02	.02	.01	0.00
8	.02	.01	.01	0.00	.06	.04	.04	.01	0.00
9	.00	0.00	.00	0.00	.01	.01	.01	.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	.05	.03	.02	0.00	.08	.10	.15	.14	.02
24	.04	.02	.02	0.00	.08	.08	.13	.11	.02
25	.07	.03	.04	0.00	.13	.11	.15	.12	.02
26	.17	.14	.09	0.00	.21	.23	.32	.28	.05
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.01	.01	.00	0.00	.00	.01	.02	.02	.00
29	.01	.01	.00	0.00	.00	.01	.02	.02	.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.11	.13	.05	0.00	.14	.18	.23	.20	.03
33	.30	.34	.14	.01	.35	.43	.54	.46	.08
34	.16	.12	.08	.00	.32	.38	.56	.48	.08
35	.80	.94	.38	.02	.70	.90	1.00	.83	.14
36	.81	.96	.38	.03	.73	.92	1.00	.83	.14
38	.81	.95	.38	.02	.71	.91	1.00	.83	.14
39	.82	1.00	.39	.10	.90	1.00	1.00	.83	.14
40	.75	.80	.35	0.00	.65	.83	1.00	.83	.14
41	.82	1.00	.39	.05	.74	.93	1.00	.83	.14
42	.82	1.00	.39	.10	.90	1.00	1.00	.83	.14
43	.82	1.00	.39	.10	.90	1.00	1.00	.83	.14
44	.82	1.00	.39	.10	.90	1.00	1.00	.83	.14
45	.82	1.00	.39	.10	.90	1.00	1.00	.83	.14
46	.66	.59	.32	0.00	.62	.74	.94	.78	.13
47	.82	.66	.39	0.00	.65	.81	1.00	.83	.14
48	.82	.98	.39	.01	.65	.86	1.00	.83	.14
49	.82	1.00	.39	.03	.69	.91	1.00	.83	.14
50	.82	1.00	.39	.05	.73	.93	1.00	.83	.14
51	.82	1.00	.39	.09	.84	.97	1.00	.83	.14
52	.64	.73	.30	.00	.52	.68	.81	.68	.11
53	.71	.83	.33	.02	.61	.78	.89	.74	.12
54	.82	1.00	.39	.06	.76	.94	1.00	.83	.14
55	.71	.83	.33	.02	.61	.78	.89	.74	.12
58	.82	1.00	.39	.09	.84	.97	1.00	.83	.14
61	.65	.74	.31	.02	.56	.72	.83	.70	.12
62	.17	.14	.09	0.00	.21	.23	.32	.28	.05
63	.17	.14	.09	0.00	.21	.23	.32	.28	.05
64	.17	.14	.09	0.00	.21	.23	.32	.28	.05
65	.17	.14	.09	0.00	.21	.23	.32	.28	.05
66	.46	.50	.22	.01	.42	.52	.63	.53	.09
67	.17	.14	.09	0.00	.21	.23	.32	.28	.05
68	.17	.14	.09	0.00	.21	.23	.32	.28	.05
69	.17	.14	.09	0.00	.21	.23	.32	.28	.05
70	.17	.14	.09	0.00	.21	.23	.32	.28	.05
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 13. 1964-1969 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	APR. 1968	MAY 1968	JUNE 1968	JULY 1968	AUG. 1968	SEP. 1968	OCT. 1968	NOV. 1968	DEC. 1968
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00	.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00	.01
24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00	.01
25	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.00	.01
26	.03	.04	.05	0.00	0.00	.02	.04	.05	.06
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.05	.08	.08	.04	.04	.06	.08	.08	.09
33	.18	.28	.29	.24	.24	.26	.28	.25	.25
34	.02	.03	.03	.03	.03	.02	.03	.05	.05
35	.51	.79	.80	.66	.64	.72	.79	.69	.70
36	.54	.85	.87	.75	.74	.80	.85	.73	.74
38	.52	.82	.83	.70	.69	.76	.82	.71	.72
39	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
40	.34	.48	.50	.26	.24	.37	.51	.53	.56
41	.63	1.00	1.00	.97	.95	1.00	1.00	.83	.82
42	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
43	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
44	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
45	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
46	.20	.27	.27	.15	.13	.21	.30	.35	.40
47	.20	.32	.32	.31	.13	.32	.32	.41	.52
48	.47	.66	.69	.44	.42	.53	.70	.68	.70
49	.63	1.00	1.00	.84	.79	.99	1.00	.83	.82
50	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
51	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
52	.34	.48	.49	.31	.29	.37	.50	.49	.51
53	.45	.67	.68	.53	.51	.60	.69	.62	.63
54	.63	1.00	1.00	.91	.89	1.00	1.00	.83	.82
55	.45	.67	.68	.53	.51	.60	.69	.62	.63
58	.63	1.00	1.00	1.00	1.00	1.00	1.00	.83	.82
61	.38	.57	.59	.44	.43	.51	.59	.54	.55
62	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
63	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
64	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
65	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
66	.24	.36	.37	.26	.25	.31	.37	.34	.35
67	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
68	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
69	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
70	.03	.04	.05	0.00	0.00	.02	.04	.06	.06
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 13. 1964-1969 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1969	FEB. 1969	MAR. 1969	APR. 1969	MAY 1969	JUNE 1969	JULY 1969	AUG. 1969	SEP. 1969	OCT. 1969
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	.00	.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	.00	.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
24	.00	.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
25	.00	.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
26	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.08	.09	.09	.09	.08	.03	.04	.04	.08	.00
33	.24	.29	.29	.29	.28	.22	.23	.25	.28	.01
34	.04	.05	.05	.05	.02	.03	.03	.03	.02	.00
35	.66	.81	.82	.82	.78	.60	.63	.67	.77	.01
36	.71	.87	.88	.87	.84	.71	.73	.76	.84	.01
38	.68	.83	.85	.84	.81	.65	.68	.71	.80	.01
39	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
40	.51	.61	.60	.60	.50	.20	.23	.28	.49	.01
41	.81	1.00	1.00	1.00	1.00	.93	.95	.98	1.00	.02
42	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
43	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
44	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
45	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
46	.34	.39	.38	.38	.30	.10	.12	.16	.29	.01
47	.37	.38	.37	.38	.32	0.00	.08	.32	.32	.01
48	.66	.80	.80	.80	.69	.36	.42	.46	.67	.01
49	.81	1.00	1.00	1.00	1.00	.72	.78	.86	1.00	.02
50	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
51	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
52	.48	.58	.58	.58	.50	.25	.29	.32	.48	.01
53	.60	.74	.73	.73	.68	.47	.50	.54	.67	.01
54	.81	1.00	1.00	1.00	1.00	.85	.88	.93	1.00	.02
55	.60	.74	.73	.73	.68	.47	.50	.54	.67	.01
58	.81	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	.02
61	.52	.64	.64	.64	.59	.39	.42	.46	.58	.01
62	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
63	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
64	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
65	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
66	.33	.40	.40	.40	.37	.23	.25	.27	.36	.01
67	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
68	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
69	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
70	.05	.06	.06	.06	.04	0.00	0.00	.00	.04	.00
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	APR. 1970	MAY 1970	JUNE 1970	JULY 1970	AUG. 1970	SEP. 1970	OCT. 1970	NOV. 1970	DEC. 1970
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
25	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
26	.01	0.00	0.00	0.00	0.00	.09	.02	.02	.03
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00	.01	.00	.00	.00
29	0.00	0.00	0.00	0.00	0.00	.01	.00	.00	.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.02	.02	.01	.01	.01	.05	.02	.02	.02
33	.11	.15	.18	.19	.19	.24	.11	.08	.08
34	.02	.01	.02	.02	.02	.09	.03	.02	.03
35	.40	.54	.57	.59	.62	.91	.41	.29	.30
36	.43	.59	.68	.69	.72	.94	.44	.30	.31
38	.42	.56	.62	.64	.67	.93	.43	.29	.31
39	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
40	.14	.14	.05	.07	.09	.49	.16	.14	.15
41	.50	.70	.88	.90	.93	1.00	.50	.33	.33
42	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
43	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
44	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
45	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
46	.08	.08	.02	.04	.04	.31	.09	.08	.09
47	.15	.06	0.00	0.00	0.00	.32	.15	.10	.10
48	.32	.39	.24	.28	.31	.81	.34	.24	.26
49	.50	.70	.73	.88	.95	1.00	.50	.33	.33
50	.50	.70	.93	.94	.97	1.00	.50	.33	.33
51	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
52	.32	.39	.24	.28	.31	.81	.34	.24	.26
53	.32	.39	.24	.28	.31	.81	.34	.24	.26
54	.47	.64	.64	.77	.83	.97	.47	.31	.32
55	.32	.39	.24	.28	.31	.81	.34	.25	.26
56	.48	.65	.79	.81	.84	1.00	.48	.32	.33
57	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
58	.50	.70	1.00	.98	1.00	1.00	.50	.33	.33
61	.32	.39	.24	.28	.31	.81	.34	.25	.26
62	.04	.04	.02	.02	.03	.15	.05	.04	.05
63	.04	.04	.02	.02	.03	.15	.05	.04	.05
64	.07	.07	.04	.05	.06	.23	.08	.07	.07
65	.07	.07	.04	.05	.06	.23	.08	.07	.07
66	.32	.39	.24	.28	.31	.81	.34	.25	.26
67	.07	.07	.04	.05	.06	.23	.08	.07	.07
68	.07	.07	.04	.05	.05	.21	.08	.06	.07
69	.07	.07	.04	.05	.06	.23	.08	.07	.07
70	.07	.07	.04	.05	.06	.23	.08	.07	.07
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	MAY 1971	JULY 1971	AUG. 1971	SEP. 1971	OCT. 1971	NOV. 1971	DEC. 1971
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	0.00	0.00	0.00	0.00	0.00
24	0.00	0.00	0.00	0.00	0.00	0.00	0.00
25	0.00	0.00	0.00	0.00	0.00	0.00	0.00
26	.00	0.00	0.00	0.00	.04	.04	.06
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	.00	.01	.01
29	0.00	0.00	0.00	0.00	.00	.01	.01
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.00	.00	.02	.03	.04	.03	.05
33	.02	.13	.21	.20	.20	.14	.21
34	.00	.02	.01	.02	.06	.05	.09
35	.08	.41	.70	.71	.71	.51	.76
36	.09	.49	.78	.77	.75	.52	.78
38	.09	.45	.74	.74	.73	.52	.77
39	.10	.74	1.00	.93	.82	.55	.81
40	.03	.03	.16	.23	.32	.26	.40
41	.10	.64	.97	.93	.82	.55	.81
42	.10	.74	1.00	.93	.82	.55	.81
43	.10	.74	1.00	.93	.82	.55	.81
44	.10	.74	1.00	.93	.82	.55	.81
45	.10	.74	1.00	.93	.82	.55	.81
46	.02	.01	.08	.13	.18	.15	.23
47	.03	0.00	0.00	.13	.26	.17	.26
48	.07	.17	.42	.53	.57	.43	.63
49	.10	.53	.99	.93	.82	.55	.81
50	.10	.68	.99	.93	.82	.55	.81
51	.10	.74	1.00	.93	.82	.55	.81
52	.07	.17	.42	.53	.57	.43	.63
53	.07	.17	.42	.53	.57	.43	.63
54	.09	.46	.88	.86	.78	.53	.79
55	.07	.17	.43	.53	.57	.43	.64
56	.10	.58	.88	.87	.80	.55	.81
57	.10	.74	1.00	.93	.82	.55	.81
58	.10	.74	1.00	.93	.82	.55	.81
61	.07	.17	.43	.53	.57	.43	.64
62	.01	.02	.04	.05	.09	.08	.12
63	.01	.02	.04	.05	.09	.08	.12
64	.02	.03	.08	.10	.15	.12	.18
65	.02	.03	.08	.10	.15	.12	.18
66	.07	.17	.42	.53	.57	.43	.63
67	.02	.03	.08	.10	.15	.12	.18
68	.02	.03	.07	.09	.13	.11	.16
69	.02	.03	.08	.10	.15	.12	.18
70	.02	.03	.08	.10	.15	.12	.18
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1976	FEB. 1976	MAR. 1976	JUNE 1976	JULY 1976	AUG. 1976	SEP. 1976	OCT. 1976
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	0.00	0.00	.00	.01	0.00	0.00
24	0.00	0.00	0.00	0.00	.00	.01	0.00	0.00
25	0.00	0.00	0.00	0.00	.00	.01	0.00	0.00
26	.02	0.00	0.00	0.00	.07	.08	0.00	0.00
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00	.00	0.00	0.00
29	0.00	0.00	0.00	0.00	0.00	.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.04	.02	.01	.05	.12	.09	0.00	0.00
33	.23	.21	.12	.19	.34	.26	.03	.03
34	.04	.01	.01	.07	.23	.20	.01	.01
35	.81	.72	.42	.61	.97	.74	.16	.16
36	.87	.79	.46	.64	.98	.77	.25	.24
38	.84	.75	.44	.62	.98	.75	.20	.19
39	1.00	.97	.56	.70	1.00	.99	.96	.93
40	.27	.17	.11	.30	.69	.53	0.00	0.00
41	1.00	.95	.55	.68	1.00	.73	.17	.16
42	1.00	.97	.56	.70	1.00	.98	.95	.92
43	1.00	.97	.56	.70	1.00	1.00	1.00	.97
44	1.00	.97	.56	.70	1.00	1.00	1.00	.97
45	1.00	.97	.56	.70	1.00	1.00	1.00	.97
46	.14	.09	.06	.16	.38	.31	0.00	0.00
47	.29	.00	.01	.18	.33	.23	0.00	0.00
48	.59	.46	.30	.41	.77	.59	0.00	0.00
49	1.00	.96	.55	.68	1.00	.69	.05	.04
50	1.00	.96	.55	.68	1.00	.73	.14	.13
51	1.00	.97	.56	.69	1.00	.88	.64	.62
52	.59	.46	.30	.41	.77	.59	0.00	0.00
53	.59	.46	.30	.41	.77	.59	0.00	0.00
54	.93	.87	.51	.63	.97	.68	.04	.03
55	.60	.46	.30	.41	.77	.59	0.00	0.00
56	.94	.87	.51	.65	1.00	.78	.31	.30
57	1.00	.97	.56	.69	1.00	.88	.64	.62
58	1.00	.97	.56	.69	1.00	.88	.64	.62
61	.60	.46	.30	.41	.77	.59	0.00	0.00
62	.07	.04	.03	.04	.14	.12	0.00	0.00
63	.07	.04	.03	.04	.14	.12	0.00	0.00
64	.13	.09	.06	.08	.21	.18	0.00	0.00
65	.13	.09	.06	.08	.21	.18	0.00	0.00
66	.60	.46	.30	.41	.77	.59	0.00	0.00
67	.13	.09	.06	.08	.21	.18	0.00	0.00
68	.12	.08	.05	.07	.19	.17	0.00	0.00
69	.13	.09	.06	.08	.21	.18	0.00	0.00
70	.13	.09	.06	.08	.21	.18	0.00	0.00
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1977	FEB. 1977	MAR. 1977	APR. 1977	MAY 1977	JUNE 1977	JULY 1977	AUG. 1977	SEP. 1977	OCT. 1977	NOV. 1977	DEC. 1977
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	0.00	0.00	.01	.00	0.00	0.00	0.00	0.00	0.00	0.00
24	0.00	0.00	0.00	0.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
25	0.00	0.00	0.00	0.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
26	0.00	0.00	0.00	0.00	.04	.01	0.00	0.00	0.00	0.00	0.00	0.00
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
29	0.00	0.00	0.00	0.00	.00	.00	0.00	0.00	0.00	0.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.00	.02	.02	.02	.06	.03	.01	.02	.02	.02	.02	.02
33	.06	.20	.19	.20	.27	.22	.20	.21	.21	.22	.22	.20
34	.01	.02	.02	.01	.13	.04	.02	.02	.02	.01	.01	.01
35	.19	.65	.61	.70	.81	.72	.64	.69	.70	.76	.77	.70
36	.22	.74	.69	.77	.86	.80	.73	.78	.79	.83	.84	.78
38	.21	.69	.65	.73	.83	.76	.68	.73	.74	.80	.80	.74
39	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
40	.03	.13	.13	.20	.36	.17	.07	.11	.13	.17	.19	.21
41	.29	.92	.86	.94	.97	.96	.92	.95	.96	.99	.99	.97
42	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
43	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
44	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
45	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
46	.01	.06	.06	.11	.19	.09	.03	.06	.06	.09	.10	.12
47	0.00	0.00	0.00	.00	.12	.03	0.00	.00	.00	.01	.02	0.00
48	.09	.36	.35	.49	.54	.43	.29	.37	.39	.51	.54	.52
49	.30	.95	.88	.95	.99	.99	.89	.98	.98	1.00	1.00	.99
50	.30	.95	.88	.95	.99	.99	.96	.98	.98	1.00	1.00	.99
51	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
52	.09	.36	.35	.49	.54	.43	.29	.37	.39	.51	.54	.52
53	.09	.36	.35	.49	.54	.43	.29	.37	.39	.51	.54	.52
54	.26	.84	.78	.87	.90	.88	.78	.86	.87	.91	.91	.90
55	.10	.37	.36	.50	.55	.44	.30	.38	.40	.51	.54	.52
56	.26	.84	.78	.88	.91	.88	.82	.86	.87	.91	.92	.92
57	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
58	.31	.97	.90	.96	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00
61	.10	.37	.36	.50	.55	.44	.30	.38	.40	.51	.54	.52
62	.01	.03	.03	.04	.08	.05	.03	.03	.04	.05	.05	.05
63	.01	.03	.03	.04	.08	.05	.03	.03	.04	.05	.05	.05
64	.02	.07	.07	.09	.14	.09	.06	.07	.08	.10	.10	.10
65	.02	.07	.07	.09	.14	.09	.06	.07	.08	.10	.10	.10
66	.10	.36	.35	.49	.55	.44	.30	.38	.40	.51	.54	.52
67	.02	.07	.07	.09	.14	.09	.06	.07	.08	.10	.10	.10
68	.02	.06	.06	.08	.12	.08	.05	.06	.07	.09	.09	.09
69	.02	.07	.07	.09	.14	.09	.05	.07	.08	.10	.10	.10
70	.02	.07	.07	.09	.14	.09	.06	.07	.08	.10	.10	.10
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1978	FEB. 1978	MAR. 1978	APR. 1978	MAY 1978	JUNE 1978	JULY 1978	AUG. 1978	SEP. 1978	OCT. 1978	NOV. 1978	DEC. 1978
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
8	0.00	0.00	.01	.02	.02	0.00	0.00	.01	.02	.03	.03	.01
9	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	0.00	0.00	.01	.02	.02	0.00	0.00	.01	.02	.03	.03	.01
24	0.00	0.00	.01	.02	.02	0.00	0.00	.01	.02	.03	.03	.01
25	0.00	0.00	.01	.02	.02	0.00	0.00	.01	.02	.03	.03	.01
26	0.00	0.00	.03	.08	.07	.01	0.00	.05	.06	.11	.11	.08
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.02	.02	.03	.10	.11	.09	.08	.11	.10	.12	.12	.12
33	.20	.20	.14	.29	.34	.31	.30	.33	.33	.37	.37	.35
34	.01	.01	.08	.22	.23	.15	.12	.21	.22	.30	.30	.25
35	.68	.67	.43	.82	.97	.93	.92	.96	.95	1.00	1.00	.98
36	.76	.76	.49	.84	.98	.95	.95	.97	.97	1.00	1.00	.99
38	.72	.71	.45	.83	.97	.94	.94	.97	.96	1.00	1.00	.99
39	1.00	1.00	.98	.99	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
40	.19	.19	.22	.61	.69	.50	.46	.66	.67	.78	.77	.71
41	.96	.96	.52	.83	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
42	1.00	1.00	.98	.99	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
43	1.00	1.00	1.00	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
44	1.00	1.00	1.00	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
45	1.00	1.00	1.00	1.00	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
46	.11	.10	.17	.42	.46	.25	.23	.41	.45	.58	.57	.42
47	0.00	0.00	.14	.27	.33	.33	.23	.33	.33	.43	.41	.33
48	.48	.47	.29	.70	.80	.62	.60	.76	.78	.88	.88	.80
49	.98	.98	.44	.80	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
50	.98	.98	.50	.82	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
51	1.00	1.00	.80	.92	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
52	.48	.47	.29	.70	.80	.62	.60	.76	.78	.88	.88	.80
53	.48	.47	.29	.70	.80	.62	.60	.76	.78	.88	.88	.80
54	.89	.89	.41	.79	.97	.93	.93	.96	.97	.98	.98	.97
55	.49	.48	.30	.70	.81	.63	.61	.77	.79	.89	.89	.81
56	.91	.91	.59	.86	1.00	.95	.95	.99	.99	1.00	1.00	1.00
57	1.00	1.00	.80	.92	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
58	1.00	1.00	.80	.92	1.00	.99	1.00	1.00	1.00	1.00	1.00	1.00
61	.49	.48	.30	.70	.81	.63	.61	.77	.79	.89	.89	.81
62	.04	.04	.05	.14	.14	.06	.05	.12	.13	.18	.18	.15
63	.04	.04	.05	.14	.14	.06	.05	.12	.13	.18	.18	.15
64	.09	.09	.08	.20	.21	.13	.12	.19	.20	.26	.26	.22
65	.09	.09	.08	.20	.21	.13	.12	.19	.20	.26	.26	.22
66	.49	.48	.29	.70	.81	.62	.60	.76	.79	.88	.88	.80
67	.09	.09	.08	.20	.21	.13	.12	.19	.20	.26	.26	.22
68	.08	.08	.08	.19	.20	.11	.10	.18	.19	.25	.24	.21
69	.09	.09	.08	.20	.21	.13	.12	.19	.20	.26	.26	.22
70	.09	.09	.08	.20	.21	.13	.12	.19	.20	.26	.26	.22
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 14. 1970-1979 EXPOSURE INDEX FOR MONTHS WHEN WELLS G OR H WERE PUMPING

MONTH YEAR NODE	JAN. 1979	FEB. 1979	MAR. 1979	APR. 1979	MAY 1979
1	0.00	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00	0.00
4	0.00	0.00	0.00	0.00	0.00
5	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00
7	0.00	0.00	0.00	0.00	0.00
8	.01	.01	0.00	.00	0.00
9	0.00	0.00	0.00	0.00	0.00
10	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00
23	.02	.01	0.00	.00	.01
24	.02	.01	0.00	.00	.00
25	.02	.01	0.00	.00	.00
26	.08	.04	.04	.07	.05
27	0.00	0.00	0.00	0.00	0.00
28	0.00	0.00	0.00	0.00	0.00
29	0.00	0.00	0.00	0.00	0.00
30	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00
32	.12	.10	.11	.13	.09
33	.35	.32	.33	.36	.25
34	.25	.20	.20	.25	.18
35	.98	.95	.95	.98	.69
36	.98	.97	.97	.99	.69
38	.98	.96	.96	.99	.69
39	1.00	1.00	1.00	1.00	.70
40	.71	.64	.59	.70	.50
41	1.00	1.00	1.00	1.00	.70
42	1.00	1.00	1.00	1.00	.70
43	1.00	1.00	1.00	1.00	.70
44	1.00	1.00	1.00	1.00	.70
45	1.00	1.00	1.00	1.00	.70
46	.45	.42	.30	.38	.27
47	.33	.33	.33	.33	.23
48	.80	.75	.69	.76	.54
49	1.00	1.00	1.00	1.00	.70
50	1.00	1.00	1.00	1.00	.70
51	1.00	1.00	1.00	1.00	.70
52	.80	.75	.69	.76	.54
53	.80	.75	.69	.76	.54
54	.97	.96	.95	.96	.68
55	.81	.76	.69	.77	.55
56	.99	.99	.98	.99	.70
57	1.00	1.00	1.00	1.00	.70
58	1.00	1.00	1.00	1.00	.70
61	.81	.76	.69	.77	.55
62	.14	.11	.10	.14	.10
63	.14	.11	.10	.14	.10
64	.22	.18	.16	.21	.15
65	.22	.18	.16	.21	.15
66	.81	.76	.69	.77	.54
67	.22	.18	.16	.21	.15
68	.20	.17	.15	.19	.14
69	.22	.18	.16	.21	.15
70	.22	.18	.16	.21	.15
71	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00

TABLE 15. 1964-1969 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH NODE	OCT. 1964	NOV. 1964	DEC. 1964	DEC. 1965
1	.00	.00	.01	.01
2	.00	.00	.01	.01
3	.00	.00	.01	.01
4	.00	.00	.01	.01
5	.00	.00	.01	.01
6	.00	.00	.00	.01
7	.01	.02	.03	.07
8	.02	.04	.06	.12
9	.00	.01	.02	.03
10	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00
23	.05	.12	.19	.29
24	.04	.09	.14	.24
25	.05	.14	.24	.45
26	.19	.40	.62	.91
27	0.00	0.00	0.00	0.00
28	.01	.02	.04	.06
29	.01	.02	.04	.06
30	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00
32	.13	.25	.37	.53
33	.36	.67	1.01	1.42
34	.18	.38	.60	.92
35	.96	1.74	2.61	3.57
36	.97	1.76	2.62	3.59
38	.97	1.75	2.62	3.58
39	.99	1.78	2.64	3.61
40	.89	1.64	2.46	3.42
41	.99	1.78	2.64	3.61
42	.99	1.78	2.64	3.61
43	.99	1.78	2.64	3.61
44	.99	1.78	2.64	3.61
45	.99	1.78	2.64	3.61
46	.77	1.44	2.16	3.08
47	.99	1.77	2.63	3.59
48	.99	1.78	2.64	3.61
49	.99	1.78	2.64	3.61
50	.99	1.78	2.64	3.61
51	.99	1.78	2.64	3.61
52	.76	1.38	2.06	2.84
53	.85	1.54	2.29	3.14
54	.99	1.78	2.64	3.61
55	.85	1.54	2.29	3.14
58	.99	1.78	2.64	3.61
61	.77	1.41	2.11	2.90
62	.19	.40	.62	.91
63	.19	.40	.62	.91
64	.19	.40	.62	.91
65	.19	.40	.62	.91
66	.54	1.01	1.51	2.11
67	.19	.40	.62	.91
68	.19	.40	.62	.91
69	.19	.40	.62	.91
70	.19	.40	.62	.91
71	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00

TABLE 15. 1964-1969 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1966	FEB. 1966	MAR. 1966	APR. 1966	MAY 1966	JUNE 1966	JULY 1966	AUG. 1966	SEP. 1966	OCT. 1966	NOV. 1966	DEC. 1966
1	.02	.02	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
2	.02	.02	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
3	.02	.02	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
4	.02	.02	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
5	.02	.02	.03	.03	.03	.03	.03	.03	.03	.03	.03	.03
6	.01	.01	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.11	.14	.17	.17	.17	.17	.17	.17	.17	.17	.17	.18
8	.18	.23	.29	.30	.33	.34	.34	.34	.35	.36	.37	.39
9	.05	.07	.08	.08	.08	.08	.08	.08	.08	.08	.08	.09
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	.39	.48	.56	.59	.63	.64	.64	.64	.66	.70	.72	.78
24	.34	.43	.51	.54	.58	.59	.59	.59	.61	.63	.65	.70
25	64	.83	1.02	1.05	1.12	1.13	1.13	1.13	1.15	1.20	1.23	1.32
26	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.07	.09	.10	.10	.10	.10	.10	.10	.10	.11	.11	.12
29	.07	.09	.10	.10	.10	.10	.10	.10	.10	.11	.11	.12
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	.68	.83	.96	1.07	1.18	1.27	1.31	1.38	1.49	1.62	1.69	1.81
33	1.84	2.25	2.63	2.95	3.25	3.54	3.78	4.04	4.36	4.71	4.90	5.21
34	1.22	1.50	1.73	1.83	1.94	1.99	2.02	2.04	2.12	2.27	2.35	2.53
35	4.57	5.57	6.57	7.48	8.35	9.16	9.81	10.52	11.42	12.37	12.90	13.71
36	4.59	5.59	6.58	7.52	8.42	9.29	10.04	10.83	11.76	12.73	13.28	14.09
38	4.58	5.58	6.58	7.50	8.38	9.22	9.92	10.66	11.58	12.54	13.08	13.89
39	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
40	4.41	5.39	6.35	7.17	7.99	8.63	8.90	9.32	10.07	10.92	11.41	12.18
41	4.61	5.61	6.61	7.61	8.55	9.55	10.52	11.52	12.52	13.52	14.09	14.91
42	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
43	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
44	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
45	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
46	4.03	4.98	5.92	6.60	7.30	7.78	7.94	8.21	8.77	9.44	9.82	10.52
47	4.59	5.59	6.59	7.25	7.93	8.48	8.75	9.08	9.74	10.48	10.93	11.75
48	4.61	5.61	6.61	7.59	8.54	9.37	9.83	10.41	11.34	12.34	12.91	13.73
49	4.61	5.61	6.61	7.61	8.55	9.55	10.41	11.41	12.41	13.41	13.97	14.80
50	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
51	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
52	3.64	4.43	5.22	5.96	6.67	7.28	7.60	8.01	8.70	9.45	9.88	10.52
53	4.02	4.90	5.77	6.61	7.41	8.16	8.70	9.33	10.14	10.99	11.47	12.18
54	4.61	5.61	6.61	7.61	8.55	9.55	10.47	11.47	12.47	13.47	14.04	14.86
55	4.02	4.90	5.77	6.61	7.41	8.16	8.70	9.33	10.14	10.99	11.47	12.18
58	4.61	5.61	6.61	7.61	8.55	9.55	10.55	11.55	12.55	13.55	14.12	14.94
61	3.72	4.53	5.33	6.08	6.79	7.45	7.90	8.44	9.15	9.91	10.34	11.00
62	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
63	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
64	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
65	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
66	2.71	3.31	3.90	4.41	4.90	5.32	5.59	5.92	6.39	6.91	7.20	7.67
67	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
68	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
69	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
70	1.20	1.48	1.74	1.89	2.05	2.11	2.11	2.14	2.25	2.41	2.51	2.70
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 15. 1964-1969 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1967	FEB. 1967	MAR. 1967	JULY 1967	AUG. 1967	SEP. 1967	OCT. 1967	NOV. 1967	DEC. 1967
1	.03	.03	.03	.03	.03	.03	.04	.04	.04
2	.03	.03	.03	.03	.03	.03	.04	.04	.04
3	.03	.03	.03	.03	.03	.03	.04	.04	.04
4	.03	.03	.03	.03	.03	.03	.04	.04	.04
5	.03	.03	.03	.03	.03	.03	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.19	.19	.20	.20	.22	.24	.26	.27	.27
8	.41	.43	.44	.44	.49	.53	.57	.58	.58
9	.09	.09	.09	.09	.10	.11	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	.83	.86	.89	.89	.97	1.07	1.22	1.36	1.39
24	.74	.76	.78	.78	.86	.94	1.07	1.18	1.19
25	1.39	1.42	1.46	1.46	1.59	1.70	1.85	1.97	1.99
26	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.13	.13	.14	.14	.14	.15	.16	.18	.19
29	.13	.13	.14	.14	.14	.15	.16	.18	.19
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	1.92	2.05	2.10	2.10	2.24	2.42	2.65	2.85	2.88
33	5.51	5.85	5.99	6.00	6.35	6.78	7.31	7.77	7.85
34	2.69	2.81	2.89	2.89	3.21	3.60	4.15	4.63	4.70
35	14.52	15.45	15.83	15.85	16.55	17.45	18.45	19.29	19.43
36	14.90	15.86	16.24	16.27	17.00	17.92	18.92	19.75	19.89
38	14.70	15.64	16.02	16.05	16.76	17.67	18.67	19.50	19.64
39	15.77	16.77	17.15	17.25	18.15	19.15	20.15	20.99	21.12
40	12.92	13.75	14.08	14.08	14.72	15.55	16.55	17.39	17.53
41	15.74	16.74	17.12	17.17	17.91	18.84	19.84	20.68	20.82
42	15.77	16.77	17.15	17.25	18.15	19.15	20.15	20.99	21.12
43	15.77	16.77	17.15	17.25	18.15	19.15	20.15	20.99	21.12
44	15.77	16.77	17.15	17.25	18.15	19.15	20.15	20.99	21.12
45	15.77	16.77	17.15	17.25	18.15	19.15	20.15	20.99	21.12
46	11.18	11.76	12.08	12.08	12.70	13.44	14.38	15.16	15.29
47	12.57	13.23	13.61	13.61	14.26	15.07	16.07	16.90	17.04
48	14.55	15.53	15.91	15.92	16.57	17.43	18.43	19.26	19.40
49	15.62	16.62	17.01	17.04	17.73	18.64	19.64	20.48	20.61
50	15.77	16.77	17.15	17.20	17.93	18.87	19.87	20.70	20.84
51	15.77	16.77	17.15	17.24	18.08	19.04	20.04	20.88	21.02
52	11.16	11.89	12.19	12.19	12.71	13.39	14.20	14.88	14.99
53	12.89	13.72	14.06	14.08	14.69	15.47	16.35	17.10	17.22
54	15.69	16.69	17.07	17.13	17.88	18.82	19.82	20.65	20.79
55	12.89	13.72	14.06	14.08	14.69	15.47	16.35	17.10	17.22
58	15.77	16.77	17.15	17.24	18.08	19.04	20.04	20.88	21.02
61	11.64	12.38	12.69	12.71	13.27	13.99	14.81	15.51	15.63
62	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
63	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
64	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
65	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
66	8.13	8.63	8.84	8.85	9.28	9.80	10.42	10.95	11.04
67	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
68	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
69	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
70	2.87	3.01	3.09	3.09	3.30	3.53	3.85	4.12	4.17
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 15. 1964-1969 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	APR. 1968	MAY 1968	JUNE 1968	JULY 1968	AUG. 1968	SEP. 1968	OCT. 1968	NOV. 1968	DEC. 1968
1	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.58	.58	.58	.58	.58	.58	.58	.58	.58
9	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.39	1.39	1.39	1.39	1.39	1.39	1.39	1.39	1.40
24	1.19	1.19	1.19	1.19	1.19	1.19	1.19	1.20	1.20
25	1.99	1.99	1.99	1.99	1.99	1.99	1.99	1.99	1.99
26	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.19	.19	.19	.19	.19	.19	.19	.19	.19
29	.19	.19	.19	.19	.19	.19	.19	.19	.19
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	2.94	3.01	3.10	3.14	3.17	3.23	3.31	3.39	3.48
33	8.03	8.31	8.59	8.84	9.07	9.33	9.62	9.86	10.12
34	4.72	4.75	4.78	4.81	4.83	4.86	4.89	4.93	4.99
35	19.93	20.72	21.52	22.18	22.82	23.54	24.33	25.01	25.71
36	20.44	21.29	22.15	22.91	23.64	24.44	25.30	26.03	26.77
38	20.17	20.99	21.82	22.52	23.20	23.96	24.78	25.49	26.20
39	21.76	22.76	23.76	24.76	25.76	26.75	27.75	28.58	29.40
40	17.86	18.35	18.84	19.11	19.34	19.71	20.22	20.76	21.32
41	21.45	22.45	23.45	24.42	25.37	26.37	27.37	28.20	29.02
42	21.76	22.76	23.76	24.76	25.76	26.75	27.75	28.58	29.40
43	21.76	22.76	23.76	24.76	25.76	26.75	27.75	28.58	29.40
44	21.76	22.76	23.76	24.76	25.76	26.75	27.75	28.58	29.40
45	21.76	22.76	23.76	24.76	25.76	26.75	27.75	28.58	29.40
46	15.49	15.76	16.02	16.17	16.30	16.51	16.80	17.16	17.56
47	17.24	17.57	17.89	18.20	18.33	18.65	18.98	19.39	19.91
48	19.87	20.53	21.21	21.66	22.07	22.60	23.30	23.98	24.68
49	21.25	22.25	23.25	24.08	24.87	25.87	26.87	27.69	28.52
50	21.47	22.47	23.47	24.47	25.47	26.47	27.47	28.30	29.12
51	21.65	22.65	23.65	24.65	25.65	26.65	27.65	28.47	29.30
52	15.33	15.80	16.30	16.61	16.90	17.27	17.78	18.27	18.78
53	17.66	18.33	19.01	19.54	20.05	20.65	21.33	21.95	22.58
54	21.43	22.43	23.43	24.34	25.22	26.22	27.22	28.04	28.87
55	17.66	18.33	19.01	19.54	20.05	20.65	21.33	21.95	22.58
58	21.65	22.65	23.65	24.65	25.65	26.65	27.65	28.47	29.30
61	16.01	16.58	17.17	17.61	18.04	18.55	19.14	19.68	20.22
62	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
63	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
64	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
65	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
66	11.29	11.64	12.01	12.27	12.53	12.84	13.21	13.55	13.90
67	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
68	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
69	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
70	4.20	4.24	4.29	4.29	4.29	4.31	4.35	4.41	4.47
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 15. 1964-1969 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1969	FEB. 1969	MAR. 1969	APR. 1969	MAY 1969	JUNE 1969	JULY 1969	AUG. 1969	SEP. 1969	OCT. 1969
1	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.40	1.40	1.40	1.41	1.41	1.41	1.41	1.41	1.41	1.41
24	1.20	1.21	1.21	1.21	1.21	1.21	1.21	1.21	1.21	1.21
25	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00
26	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.19	.19	.19	.19	.19	.19	.19	.19	.19	.19
29	.19	.19	.19	.19	.19	.19	.19	.19	.19	.19
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	3.55	3.64	3.74	3.83	3.91	3.94	3.97	4.01	4.09	4.09
33	10.35	10.64	10.94	11.23	11.51	11.73	11.96	12.21	12.48	12.49
34	5.02	5.07	5.12	5.17	5.19	5.22	5.24	5.27	5.28	5.29
35	26.37	27.17	27.99	28.81	29.59	30.19	30.82	31.49	32.26	32.27
36	27.47	28.34	29.21	30.09	30.93	31.64	32.37	33.13	33.97	33.99
38	26.88	27.72	28.56	29.41	30.21	30.87	31.54	32.26	33.06	33.07
39	30.22	31.22	32.22	33.22	34.22	35.22	36.22	37.22	38.22	38.23
40	21.82	22.43	23.03	23.62	24.13	24.33	24.56	24.84	25.33	25.34
41	29.83	30.83	31.83	32.83	33.83	34.76	35.71	36.69	37.69	37.70
42	30.22	31.22	32.22	33.22	34.22	35.22	36.22	37.22	38.22	38.23
43	30.22	31.22	32.22	33.22	34.22	35.22	36.22	37.22	38.22	38.23
44	30.22	31.22	32.22	33.22	34.22	35.22	36.22	37.22	38.22	38.23
45	30.22	31.22	32.22	33.22	34.22	35.22	36.22	37.22	38.22	38.23
46	17.90	18.29	18.67	19.05	19.35	19.45	19.57	19.72	20.01	20.02
47	20.27	20.65	21.03	21.41	21.73	21.73	21.81	22.13	22.45	22.46
48	25.34	26.14	26.94	27.74	28.43	28.79	29.21	29.67	30.34	30.35
49	29.33	30.33	31.33	32.33	33.33	34.05	34.83	35.69	36.69	36.71
50	29.93	30.93	31.93	32.93	33.93	34.93	35.93	36.93	37.93	37.95
51	30.11	31.11	32.11	33.11	34.11	35.11	36.11	37.11	38.11	38.12
52	19.26	19.84	20.42	20.99	21.49	21.75	22.04	22.36	22.84	22.85
53	23.18	23.92	24.65	25.38	26.07	26.54	27.04	27.58	28.25	28.27
54	29.68	30.68	31.68	32.68	33.68	34.53	35.40	36.33	37.33	37.34
55	23.18	23.92	24.65	25.38	26.07	26.54	27.04	27.58	28.25	28.27
58	30.11	31.11	32.11	33.11	34.11	35.11	36.11	37.11	38.11	38.12
61	20.75	21.38	22.02	22.65	23.24	23.63	24.06	24.51	25.09	25.10
62	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
63	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
64	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
65	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
66	14.23	14.64	15.04	15.44	15.81	16.04	16.30	16.57	16.93	16.93
67	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
68	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
69	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
70	4.52	4.58	4.64	4.69	4.74	4.74	4.74	4.74	4.78	4.78
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	APR. 1970	MAY 1970	JUNE 1970	JULY 1970	AUG. 1970	SEP. 1970	OCT. 1970	NOV. 1970	DEC. 1970
1	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.41	1.41	1.41	1.41	1.41	1.41	1.41	1.41	1.41
24	1.21	1.21	1.21	1.21	1.21	1.21	1.21	1.21	1.21
25	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00
26	4.79	4.79	4.79	4.79	4.79	4.88	4.90	4.93	4.95
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.19	.19	.19	.19	.19	.20	.20	.20	.20
29	.19	.19	.19	.19	.19	.20	.20	.20	.20
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	4.11	4.12	4.13	4.14	4.15	4.20	4.22	4.23	4.25
33	12.60	12.75	12.93	13.12	13.31	13.56	13.67	13.75	13.83
34	5.31	5.32	5.34	5.36	5.38	5.48	5.50	5.52	5.55
35	32.67	33.21	33.78	34.37	35.00	35.91	36.32	36.61	36.91
36	34.42	35.01	35.69	36.38	37.10	38.05	38.49	38.79	39.10
38	33.49	34.05	34.67	35.31	35.98	36.90	37.33	37.62	37.93
39	38.73	39.43	40.43	41.42	42.42	43.42	43.92	44.24	44.58
40	25.48	25.62	25.67	25.74	25.83	26.32	26.48	26.61	26.76
41	38.20	38.90	39.78	40.67	41.60	42.60	43.10	43.43	43.76
42	38.73	39.43	40.43	41.42	42.42	43.42	43.92	44.24	44.58
43	38.73	39.43	40.43	41.42	42.42	43.42	43.92	44.24	44.58
44	38.73	39.43	40.43	41.42	42.42	43.42	43.92	44.24	44.58
45	38.73	39.43	40.43	41.42	42.42	43.42	43.92	44.24	44.58
46	20.10	20.18	20.20	20.23	20.28	20.59	20.68	20.76	20.85
47	22.61	22.67	22.67	22.67	22.67	22.99	23.14	23.24	23.34
48	30.67	31.05	31.29	31.57	31.87	32.68	33.01	33.26	33.52
49	37.21	37.91	38.64	39.52	40.47	41.47	41.97	42.30	42.63
50	38.45	39.15	40.08	41.02	41.99	42.99	43.49	43.82	44.15
51	38.62	39.32	40.32	41.31	42.31	43.31	43.81	44.13	44.47
52	23.17	23.55	23.79	24.07	24.37	25.18	25.51	25.76	26.02
53	28.59	28.97	29.21	29.49	29.79	30.60	30.93	31.18	31.44
54	37.81	38.45	39.09	39.86	40.68	41.65	42.13	42.44	42.76
55	28.59	28.98	29.21	29.50	29.81	30.62	30.96	31.21	31.47
56	37.82	38.47	39.26	40.07	40.90	41.90	42.38	42.70	43.03
57	38.62	39.32	40.32	41.31	42.31	43.31	43.81	44.13	44.47
58	38.62	39.32	40.32	41.31	42.31	43.31	43.81	44.13	44.47
61	25.42	25.81	26.04	26.33	26.64	27.45	27.79	28.04	28.30
62	4.82	4.86	4.88	4.90	4.93	5.08	5.13	5.17	5.22
63	4.82	4.86	4.88	4.90	4.93	5.08	5.13	5.17	5.22
64	4.85	4.93	4.97	5.02	5.08	5.31	5.40	5.46	5.53
65	4.85	4.93	4.97	5.02	5.08	5.31	5.40	5.46	5.53
66	17.25	17.64	17.87	18.15	18.46	19.27	19.61	19.85	20.11
67	4.85	4.93	4.97	5.02	5.08	5.31	5.40	5.46	5.53
68	4.85	4.91	4.95	5.00	5.05	5.26	5.34	5.40	5.46
69	4.85	4.93	4.97	5.02	5.08	5.31	5.40	5.46	5.53
70	4.85	4.93	4.97	5.02	5.08	5.31	5.40	5.46	5.53
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH / YEAR / NODE	MAY 1971	JULY 1971	AUG. 1971	SEP. 1971	OCT. 1971	NOV. 1971	DEC. 1971
1	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.41	1.41	1.41	1.41	1.41	1.41	1.41
24	1.21	1.21	1.21	1.21	1.21	1.21	1.21
25	2.00	2.00	2.00	2.00	2.00	2.00	2.00
26	4.96	4.96	4.96	4.96	5.00	5.04	5.10
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.20	.20	.20	.20	.20	.21	.22
29	.20	.20	.20	.20	.20	.21	.22
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	4.25	4.26	4.28	4.30	4.34	4.38	4.43
33	13.85	13.98	14.19	14.39	14.59	14.73	14.95
34	5.55	5.57	5.58	5.60	5.66	5.71	5.81
35	36.99	37.40	38.10	38.81	39.52	40.03	40.79
36	39.19	39.68	40.46	41.24	41.99	42.51	43.29
38	38.01	38.46	39.20	39.94	40.67	41.18	41.95
39	44.68	45.41	46.41	47.34	48.16	48.71	49.53
40	26.79	26.82	26.98	27.21	27.52	27.78	28.18
41	43.86	44.50	45.46	46.39	47.21	47.76	48.57
42	44.68	45.41	46.41	47.34	48.16	48.71	49.53
43	44.68	45.41	46.41	47.34	48.16	48.71	49.53
44	44.68	45.41	46.41	47.34	48.16	48.71	49.53
45	44.68	45.41	46.41	47.34	48.16	48.71	49.53
46	20.86	20.88	20.96	21.08	21.26	21.41	21.64
47	23.37	23.37	23.37	23.50	23.76	23.93	24.19
48	33.58	33.75	34.17	34.70	35.27	35.70	36.33
49	42.73	43.26	44.24	45.17	45.99	46.54	47.36
50	44.25	44.93	45.92	46.84	47.67	48.22	49.03
51	44.57	45.30	46.30	47.23	48.05	48.60	49.42
52	26.08	26.25	26.67	27.20	27.77	28.20	28.83
53	31.50	31.67	32.09	32.62	33.19	33.62	34.25
54	42.85	43.32	44.20	45.06	45.84	46.37	47.15
55	31.53	31.70	32.13	32.66	33.24	33.67	34.30
56	43.13	43.70	44.59	45.46	46.26	46.81	47.61
57	44.57	45.30	46.30	47.23	48.05	48.60	49.42
58	44.57	45.30	46.30	47.23	48.05	48.60	49.42
61	28.36	28.53	28.96	29.49	30.07	30.50	31.14
62	5.23	5.25	5.28	5.33	5.42	5.50	5.62
63	5.23	5.25	5.28	5.33	5.42	5.50	5.62
64	5.55	5.58	5.66	5.76	5.91	6.03	6.20
65	5.55	5.58	5.66	5.76	5.91	6.03	6.20
66	20.18	20.35	20.77	21.30	21.87	22.30	22.93
67	5.55	5.58	5.66	5.76	5.91	6.03	6.20
68	5.48	5.50	5.57	5.66	5.80	5.91	6.07
69	5.55	5.58	5.66	5.76	5.91	6.03	6.20
70	5.55	5.58	5.66	5.76	5.91	6.03	6.20
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1972	FEB. 1972	MAR. 1972	APR. 1972	AUG. 1974	SEP. 1974	OCT. 1974	NOV. 1974	DEC. 1974
1	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.41	1.41	1.41	1.41	1.41	1.46	1.51	1.57	1.58
24	1.21	1.21	1.21	1.21	1.21	1.24	1.27	1.31	1.32
25	2.00	2.00	2.00	2.00	2.00	2.03	2.06	2.10	2.11
26	5.15	5.22	5.27	5.30	5.30	5.44	5.59	5.73	5.79
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.22	.22	.23	.23	.23	.24	.26	.27	.28
29	.22	.22	.23	.23	.23	.24	.26	.27	.28
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	4.47	4.52	4.57	4.59	4.61	4.76	4.92	5.04	5.08
33	15.15	15.40	15.62	15.75	15.95	16.36	16.80	17.19	17.33
34	5.86	5.95	6.02	6.05	6.09	6.50	6.95	7.30	7.40
35	41.53	42.43	43.23	43.70	44.31	45.28	46.27	47.19	47.56
36	44.05	44.99	45.83	46.32	47.02	48.00	48.99	49.93	50.32
38	42.70	43.62	44.44	44.92	45.57	46.55	47.53	48.47	48.85
39	50.36	51.36	52.27	52.81	53.78	54.78	55.78	56.77	57.17
40	28.48	28.95	29.32	29.51	29.62	30.45	31.32	32.02	32.27
41	49.41	50.41	51.32	51.86	52.73	53.72	54.71	55.69	56.10
42	50.36	51.36	52.27	52.81	53.78	54.78	55.78	56.77	57.17
43	50.36	51.36	52.27	52.81	53.78	54.78	55.78	56.77	57.17
44	50.36	51.36	52.27	52.81	53.78	54.78	55.78	56.77	57.17
45	50.36	51.36	52.27	52.81	53.78	54.78	55.78	56.77	57.17
46	21.80	22.08	22.30	22.40	22.45	23.03	23.64	24.13	24.31
47	24.44	24.76	25.05	25.21	25.25	25.85	26.49	27.01	27.23
48	36.91	37.65	38.30	38.67	38.95	39.82	40.72	41.55	41.88
49	48.19	49.19	50.10	50.64	51.38	52.37	53.36	54.34	54.74
50	49.86	50.86	51.78	52.32	53.23	54.22	55.22	56.20	56.60
51	50.25	51.25	52.16	52.70	53.67	54.67	55.67	56.65	57.05
52	29.41	30.15	30.80	31.17	31.45	32.32	33.22	34.05	34.38
53	34.83	35.57	36.22	36.59	36.87	37.74	38.64	39.47	39.80
54	47.94	48.90	49.77	50.29	50.94	51.91	52.89	53.85	54.24
55	34.89	35.63	36.29	36.67	36.95	37.83	38.74	39.57	39.90
56	48.42	49.41	50.30	50.83	51.61	52.60	53.59	54.55	54.95
57	50.25	51.25	52.16	52.70	53.67	54.67	55.67	56.65	57.05
58	50.25	51.25	52.16	52.70	53.67	54.67	55.67	56.65	57.05
61	31.72	32.46	33.12	33.50	33.78	34.66	35.57	36.40	36.73
62	5.71	5.84	5.95	6.01	6.04	6.24	6.47	6.67	6.76
63	5.71	5.84	5.95	6.01	6.04	6.24	6.47	6.67	6.76
64	6.36	6.56	6.73	6.82	6.88	7.16	7.46	7.74	7.86
65	6.36	6.56	6.73	6.82	6.88	7.16	7.46	7.74	7.86
66	23.51	24.26	24.91	25.28	25.56	26.44	27.34	28.17	28.50
67	6.36	6.56	6.73	6.82	6.88	7.16	7.46	7.74	7.86
68	6.21	6.39	6.55	6.64	6.69	6.95	7.24	7.50	7.61
69	6.36	6.56	6.73	6.82	6.88	7.16	7.46	7.74	7.86
70	6.36	6.56	6.73	6.82	6.88	7.16	7.46	7.74	7.86
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	MAY 1975	JUNE 1975	JULY 1975	AUG. 1975	SEP. 1975	OCT. 1975	NOV. 1975	DEC. 1975
1	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.58	1.58	1.58	1.59	1.61	1.61	1.61	1.61
24	1.32	1.32	1.32	1.33	1.34	1.34	1.34	1.34
25	2.11	2.11	2.11	2.12	2.13	2.13	2.13	2.13
26	5.79	5.80	5.84	5.90	5.94	5.95	5.96	5.96
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.28	.28	.28	.28	.28	.28	.28	.28
29	.28	.28	.28	.28	.28	.28	.28	.28
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	5.09	5.12	5.22	5.31	5.37	5.39	5.41	5.43
33	17.42	17.63	17.96	18.26	18.55	18.69	18.78	19.00
34	7.40	7.44	7.64	7.82	7.98	8.00	8.01	8.03
35	47.99	48.56	49.50	50.36	51.21	51.71	52.04	52.80
36	50.68	51.44	52.40	53.30	54.20	54.74	55.09	55.91
38	49.19	49.90	50.85	51.73	52.60	53.12	53.46	54.24
39	57.64	58.64	59.64	60.64	61.64	62.26	62.68	63.64
40	32.35	32.50	33.10	33.59	34.01	34.17	34.26	34.47
41	56.55	57.49	58.48	59.46	60.45	61.08	61.50	62.46
42	57.64	58.64	59.64	60.64	61.64	62.26	62.68	63.64
43	57.64	58.64	59.64	60.64	61.64	62.26	62.68	63.64
44	57.64	58.64	59.64	60.64	61.64	62.26	62.68	63.64
45	57.64	58.64	59.64	60.64	61.64	62.26	62.68	63.64
46	24.35	24.43	24.75	25.02	25.29	25.37	25.42	25.53
47	27.23	27.25	27.57	27.76	28.03	28.21	28.32	28.55
48	42.08	42.44	43.12	43.74	44.40	44.77	45.01	45.55
49	55.20	56.18	57.17	58.16	59.15	59.78	60.19	61.15
50	57.06	58.03	59.03	60.02	61.01	61.64	62.06	63.02
51	57.52	58.52	59.52	60.52	61.52	62.14	62.56	63.52
52	34.58	34.94	35.62	36.24	36.90	37.27	37.51	38.05
53	40.00	40.36	41.04	41.66	42.32	42.69	42.93	43.47
54	54.65	55.51	56.45	57.37	58.30	58.88	59.27	60.15
55	40.11	40.47	41.16	41.79	42.45	42.83	43.07	43.61
56	55.36	56.22	57.19	58.13	59.07	59.66	60.05	60.95
57	57.52	58.52	59.52	60.52	61.52	62.14	62.56	63.52
58	57.52	58.52	59.52	60.52	61.52	62.14	62.56	63.52
61	36.94	37.30	37.99	38.62	39.28	39.66	39.90	40.44
62	6.78	6.81	6.91	7.02	7.13	7.17	7.19	7.24
63	6.78	6.81	6.91	7.02	7.13	7.17	7.19	7.24
64	7.90	7.97	8.14	8.31	8.47	8.56	8.60	8.71
65	7.90	7.97	8.14	8.31	8.47	8.56	8.60	8.71
66	28.71	29.07	29.76	30.38	31.04	31.41	31.65	32.19
67	7.90	7.97	8.14	8.31	8.47	8.56	8.60	8.71
68	7.65	7.71	7.86	8.02	8.17	8.25	8.29	8.38
69	7.90	7.97	8.14	8.31	8.47	8.56	8.60	8.71
70	7.90	7.97	8.14	8.31	8.47	8.56	8.60	8.71
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1976	FEB. 1976	MAR. 1976	JUNE 1976	JULY 1976	AUG. 1976	SEP. 1976	OCT. 1976
1	.04	.04	.04	.04	.04	04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.61	1.61	1.61	1.61	1.61	1.62	1.62	1.62
24	1.34	1.34	1.34	1.34	1.34	1.34	1.34	1.34
25	2.13	2.13	2.13	2.13	2.13	2.13	2.13	2.13
26	5.98	5.98	5.98	5.98	6.05	6.13	6.13	6.13
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.28	.28	.28	.28	.28	.29	.29	.29
29	.28	.28	.28	.28	.28	.29	.29	.29
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	5.47	5.49	5.51	5.56	5.67	5.76	5.76	5.76
33	19.23	19.44	19.56	19.76	20.10	20.36	20.39	20.41
34	8.08	8.09	8.10	8.17	8.39	8.59	8.60	8.60
35	53.61	54.33	54.75	55.36	56.33	57.07	57.23	57.39
36	56.78	57.57	58.03	58.67	59.65	60.41	60.66	60.90
38	55.09	55.84	56.28	56.90	57.88	58.63	58.82	59.02
39	64.64	65.61	66.16	66.86	67.86	68.85	69.81	70.73
40	34.74	34.91	35.02	35.32	36.01	36.55	36.55	36.55
41	63.46	64.40	64.95	65.63	66.63	67.36	67.53	67.69
42	64.64	65.61	66.16	66.86	67.86	68.84	69.80	70.72
43	64.64	65.61	66.16	66.87	67.87	68.87	69.87	70.83
44	64.64	65.61	66.16	66.87	67.87	68.87	69.87	70.83
45	64.64	65.61	66.16	66.87	67.87	68.87	69.87	70.83
46	25.67	25.75	25.81	25.97	26.35	26.66	26.66	26.66
47	28.84	28.84	28.86	29.03	29.37	29.59	29.59	29.59
48	46.15	46.60	46.90	47.31	48.08	48.67	48.67	48.67
49	62.15	63.11	63.67	64.34	65.34	66.03	66.08	66.11
50	64.02	64.98	65.53	66.20	67.20	67.93	68.07	68.20
51	64.52	65.49	66.04	66.73	67.73	68.61	69.25	69.87
52	38.65	39.10	39.40	39.81	40.58	41.17	41.17	41.17
53	44.07	44.52	44.82	45.23	46.00	46.59	46.59	46.59
54	61.08	61.95	62.45	63.08	64.05	64.72	64.76	64.79
55	44.21	44.67	44.97	45.38	45.15	46.75	46.75	46.75
56	61.89	62.76	63.27	63.92	64.92	65.69	66.00	66.30
57	64.52	65.49	66.04	66.73	67.73	68.61	69.25	69.87
58	64.52	65.49	66.04	66.73	67.73	68.61	69.25	69.87
61	41.04	41.50	41.80	42.21	42.98	43.58	43.58	43.58
62	7.31	7.35	7.38	7.42	7.56	7.68	7.68	7.68
63	7.31	7.35	7.38	7.42	7.56	7.68	7.68	7.68
64	8.84	8.92	8.98	9.06	9.27	9.45	9.45	9.45
65	8.84	8.92	8.98	9.06	9.27	9.45	9.45	9.45
66	32.79	33.25	33.55	33.96	34.73	35.32	35.32	35.32
67	8.84	8.92	8.98	9.06	9.27	9.45	9.45	9.45
68	8.50	8.57	8.62	8.69	8.89	9.05	9.05	9.05
69	8.84	8.92	8.98	9.06	9.27	9.45	9.45	9.45
70	8.84	8.92	8.98	9.06	9.27	9.45	9.45	9.45
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1977	FEB. 1977	MAR. 1977	APR. 1977	MAY 1977	JUNE 1977	JULY 1977	AUG. 1977	SEP. 1977	OCT. 1977	NOV. 1977	DEC. 1977
1	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.59	.59	.59	.59	.59	.59	.59	.59	.59	.59
9	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.62	1.62	1.62	1.62	1.62	1.63	1.63	1.63	1.63	1.63	1.63	1.63
24	1.34	1.34	1.34	1.34	1.35	1.35	1.35	1.35	1.35	1.35	1.35	1.35
25	2.13	2.13	2.13	2.13	2.14	2.14	2.14	2.14	2.14	2.14	2.14	2.14
26	6.13	6.13	6.13	6.13	6.16	6.17	6.17	6.17	6.17	6.17	6.17	6.17
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29
29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	5.77	5.78	5.80	5.82	5.88	5.91	5.92	5.93	5.95	5.97	6.00	6.02
33	20.47	20.67	20.86	21.06	21.33	21.55	21.75	21.95	22.16	22.38	22.60	22.80
34	8.61	8.63	8.64	8.65	8.79	8.83	8.85	8.87	8.88	8.90	8.91	8.92
35	57.58	58.23	58.84	59.54	60.35	61.07	61.71	62.40	63.10	63.86	64.63	65.33
36	61.13	61.87	62.56	63.33	64.19	64.99	65.73	66.50	67.29	68.12	68.96	69.74
38	59.22	59.92	60.57	61.30	62.13	62.89	63.57	64.30	65.04	65.84	66.64	67.38
39	71.04	72.01	72.91	73.87	74.87	75.87	76.86	77.86	78.86	79.86	80.86	81.86
40	36.57	36.70	36.83	37.02	37.38	37.55	37.82	37.73	37.86	38.03	38.22	38.43
41	67.98	68.90	69.76	70.70	71.66	72.62	73.54	74.49	75.45	76.44	77.43	78.39
42	71.03	72.00	72.90	73.86	74.86	75.86	76.85	77.85	78.85	79.85	80.85	81.85
43	71.14	72.11	73.01	73.97	74.97	75.97	76.96	77.96	78.96	79.96	80.96	81.96
44	71.14	72.11	73.01	73.97	74.97	75.97	76.96	77.96	78.96	79.96	80.96	81.96
45	71.14	72.11	73.01	73.97	74.97	75.97	76.96	77.96	78.96	79.96	80.96	81.96
46	26.68	26.74	26.80	26.91	27.10	27.19	27.23	27.28	27.34	27.43	27.53	27.65
47	29.59	29.59	29.59	29.60	29.72	29.74	29.74	29.75	29.75	29.76	29.78	29.78
48	48.76	49.12	49.47	49.96	50.50	50.94	51.23	51.60	52.00	52.51	53.04	53.56
49	66.41	67.36	68.24	69.20	70.19	71.17	72.06	73.04	74.03	75.02	76.02	77.00
50	68.50	69.45	70.33	71.29	72.27	73.26	74.22	75.20	76.18	77.18	78.17	79.16
51	70.18	71.15	72.05	73.01	74.01	75.01	76.00	77.00	78.00	79.00	80.00	81.00
52	41.26	41.62	41.97	42.46	43.00	43.44	43.73	44.10	44.50	45.01	45.54	46.06
53	46.68	47.04	47.39	47.88	48.42	48.86	49.15	49.52	49.92	50.43	50.96	51.48
54	65.05	65.88	66.67	67.54	68.44	69.32	70.10	70.96	71.83	72.74	73.65	74.55
55	46.84	47.21	47.57	48.06	48.61	49.05	49.35	49.73	50.13	50.65	51.18	51.70
56	66.56	67.40	68.18	69.06	69.97	70.85	71.67	72.53	73.40	74.31	75.23	76.15
57	70.18	71.15	72.05	73.01	74.01	75.01	76.00	77.00	78.00	79.00	80.00	81.00
58	70.18	71.15	72.05	73.01	74.01	75.01	76.00	77.00	78.00	79.00	80.00	81.00
61	43.67	44.04	44.40	44.89	45.44	45.88	46.18	46.56	46.96	47.48	48.01	48.53
62	7.69	7.72	7.75	7.79	7.87	7.92	7.95	7.98	8.01	8.06	8.11	8.15
63	7.69	7.72	7.75	7.79	7.87	7.92	7.95	7.98	8.01	8.06	8.11	8.15
64	9.47	9.53	9.60	9.69	9.83	9.92	9.97	10.05	10.12	10.22	10.32	10.42
65	9.47	9.53	9.60	9.69	9.83	9.92	9.97	10.05	10.12	10.22	10.32	10.42
66	35.41	35.78	36.13	36.62	37.17	37.60	37.90	38.28	38.67	39.19	39.72	40.24
67	9.47	9.53	9.60	9.69	9.83	9.92	9.97	10.05	10.12	10.22	10.32	10.42
68	9.07	9.13	9.19	9.27	9.39	9.47	9.52	9.59	9.65	9.74	9.83	9.91
69	9.47	9.53	9.60	9.69	9.83	9.92	9.97	10.05	10.12	10.22	10.32	10.42
70	9.47	9.53	9.60	9.69	9.83	9.92	9.97	10.05	10.12	10.22	10.32	10.42
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1978	FEB. 1978	MAR. 1978	APR. 1978	MAY 1978	JUNE 1978	JULY 1978	AUG. 1978	SEP. 1978	OCT. 1978	NOV. 1978	DEC. 1978
1	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27	.27
8	.59	.59	.60	.62	.64	.64	.64	.64	.66	.69	.72	.72
9	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
23	1.63	1.63	1.63	1.66	1.67	1.67	1.67	1.68	1.70	1.73	1.76	1.77
24	1.35	1.35	1.35	1.38	1.40	1.40	1.40	1.40	1.42	1.45	1.48	1.49
25	2.14	2.14	2.14	2.17	2.18	2.18	2.18	2.19	2.21	2.24	2.27	2.28
26	6.17	6.17	6.20	6.28	6.35	6.36	6.36	6.41	6.47	6.58	6.69	6.77
27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
28	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29
29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29	.29
30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
32	6.04	6.06	6.09	6.19	6.30	6.38	6.46	6.57	6.67	6.80	6.92	7.04
33	22.99	23.19	23.33	23.62	23.96	24.26	24.56	24.89	25.22	25.59	25.95	26.30
34	8.93	8.95	9.02	9.25	9.48	9.63	9.75	9.97	10.19	10.49	10.79	11.03
35	66.01	66.68	67.10	67.93	68.89	69.82	70.74	71.70	72.65	73.65	74.65	75.63
36	70.50	71.26	71.75	72.59	73.57	74.52	75.47	76.44	77.41	78.41	79.41	80.40
38	68.09	68.81	69.26	70.09	71.06	72.00	72.93	73.90	74.86	75.86	76.86	77.84
39	82.86	83.86	84.84	85.83	86.83	87.82	88.82	89.82	90.82	91.82	92.82	93.82
40	38.62	38.81	39.03	39.63	40.32	40.82	41.28	41.93	42.60	43.38	44.15	44.87
41	79.35	80.31	80.83	81.66	82.66	83.65	84.65	85.65	86.65	87.65	88.65	89.65
42	82.85	83.85	84.63	85.82	86.82	87.81	88.81	89.81	90.81	91.81	92.81	93.81
43	82.96	83.96	84.96	85.96	86.96	87.95	88.95	89.95	90.95	91.95	92.95	93.95
44	82.96	83.96	84.96	85.96	86.96	87.95	88.95	89.95	90.95	91.95	92.95	93.95
45	82.96	83.96	84.96	85.96	86.96	87.95	88.95	89.95	90.95	91.95	92.95	93.95
46	27.76	27.86	28.03	28.45	28.91	29.17	29.40	29.80	30.25	30.83	31.39	31.81
47	29.78	29.78	29.92	30.19	30.52	30.85	31.09	31.42	31.74	32.18	32.58	32.91
48	54.04	54.52	54.81	55.51	56.31	56.93	57.53	58.29	59.08	59.96	60.84	61.64
49	77.99	78.97	79.41	80.21	81.21	82.20	83.20	84.20	85.20	86.20	87.20	88.20
50	80.14	81.13	81.63	82.45	83.45	84.44	85.44	86.44	87.44	88.44	89.44	90.44
51	82.00	83.00	83.80	84.72	85.72	86.72	87.72	88.72	89.72	90.72	91.72	92.72
52	46.54	47.02	47.31	48.01	48.81	49.43	50.03	50.79	51.58	52.46	53.34	54.14
53	51.96	52.44	52.73	53.43	54.23	54.85	55.45	56.21	57.00	57.88	58.76	59.56
54	75.45	76.34	76.75	77.53	78.50	79.43	80.36	81.33	82.29	83.28	84.26	85.23
55	52.19	52.67	52.97	53.67	54.49	55.11	55.72	56.48	57.28	58.16	59.05	59.86
56	77.06	77.97	78.56	79.41	80.41	81.36	82.31	83.30	84.30	85.29	86.29	87.28
57	82.00	83.00	83.80	84.72	85.72	86.72	87.72	88.72	89.72	90.72	91.72	92.72
58	82.00	83.00	83.80	84.72	85.72	86.72	87.72	88.72	89.72	90.72	91.72	92.72
61	49.02	49.50	49.80	50.50	51.32	51.94	52.55	53.31	54.11	54.99	55.88	56.69
62	8.20	8.24	8.29	8.43	8.57	8.63	8.68	8.80	8.93	9.11	9.29	9.44
63	8.20	8.24	8.29	8.43	8.57	8.63	8.68	8.80	8.93	9.11	9.29	9.44
64	10.51	10.60	10.68	10.89	11.10	11.23	11.34	11.53	11.74	12.00	12.26	12.49
65	10.51	10.60	10.68	10.89	11.10	11.23	11.34	11.53	11.74	12.00	12.26	12.49
66	40.72	41.20	41.49	42.19	43.00	43.63	44.23	44.99	45.78	46.66	47.55	48.35
67	10.51	10.60	10.68	10.89	11.10	11.23	11.34	11.53	11.74	12.00	12.26	12.49
68	10.00	10.08	10.15	10.34	10.53	10.65	10.75	10.92	11.11	11.36	11.60	11.81
69	10.51	10.60	10.68	10.89	11.10	11.23	11.34	11.53	11.74	12.00	12.26	12.49
70	10.51	10.60	10.68	10.89	11.10	11.23	11.34	11.53	11.74	12.00	12.26	12.49
71	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 16. 1970-1979 CUMULATIVE EXPOSURE (BEFORE, AND INCLUDING, THIS MONTH)

MONTH YEAR NODE	JAN. 1979	FEB. 1979	MAR. 1979	APR. 1979	MAY 1979
1	.04	.04	.04	.04	.04
2	.04	.04	.04	.04	.04
3	.04	.04	.04	.04	.04
4	.04	.04	.04	.04	.04
5	.04	.04	.04	.04	.04
6	.02	.02	.02	.02	.02
7	.27	.27	.27	.27	.27
8	.74	.75	.75	.75	.75
9	.12	.12	.12	.12	.12
10	0.00	0.00	0.00	0.00	0.00
11	0.00	0.00	0.00	0.00	0.00
12	0.00	0.00	0.00	0.00	0.00
13	0.00	0.00	0.00	0.00	0.00
14	0.00	0.00	0.00	0.00	0.00
15	0.00	0.00	0.00	0.00	0.00
16	0.00	0.00	0.00	0.00	0.00
17	0.00	0.00	0.00	0.00	0.00
18	0.00	0.00	0.00	0.00	0.00
19	0.00	0.00	0.00	0.00	0.00
20	0.00	0.00	0.00	0.00	0.00
21	0.00	0.00	0.00	0.00	0.00
22	0.00	0.00	0.00	0.00	0.00
23	1.79	1.80	1.80	1.80	1.81
24	1.51	1.52	1.52	1.52	1.52
25	2.30	2.31	2.31	2.31	2.31
26	6.84	6.89	6.92	7.00	7.05
27	0.00	0.00	0.00	0.00	0.00
28	.29	.29	.29	.29	.29
29	.29	.29	.29	.29	.29
30	0.00	0.00	0.00	0.00	0.00
31	0.00	0.00	0.00	0.00	0.00
32	7.15	7.25	7.36	7.48	7.57
33	26.65	26.97	27.30	27.65	27.90
34	11.28	11.48	11.68	11.94	12.11
35	76.60	77.55	78.50	79.49	80.17
36	81.38	82.35	83.32	84.31	85.00
38	78.82	79.78	80.74	81.72	82.41
39	94.82	95.82	96.82	97.82	98.52
40	45.57	46.21	46.80	47.51	48.00
41	90.65	91.65	92.65	93.65	94.35
42	94.81	95.81	96.81	97.81	98.51
43	94.95	95.95	96.95	97.95	98.65
44	94.95	95.95	96.95	97.95	98.65
45	94.95	95.95	96.95	97.95	98.65
46	32.26	32.67	32.98	33.35	33.62
47	33.24	33.58	33.91	34.24	34.47
48	62.44	63.19	63.88	64.64	65.18
49	89.20	90.20	91.20	92.20	92.90
50	91.44	92.44	93.44	94.44	95.14
51	93.72	94.72	95.72	96.72	97.42
52	54.94	55.69	56.38	57.14	57.68
53	60.36	61.11	61.80	62.56	63.10
54	86.20	87.16	88.11	89.07	89.75
55	60.67	61.43	62.12	62.89	63.44
56	88.28	89.27	90.25	91.24	91.94
57	93.72	94.72	95.72	96.72	97.42
58	93.72	94.72	95.72	96.72	97.42
61	57.50	58.26	58.95	59.72	60.27
62	9.58	9.69	9.79	9.92	10.02
63	9.58	9.69	9.79	9.92	10.02
64	12.71	12.89	13.05	13.26	13.41
65	12.71	12.89	13.05	13.26	13.41
66	49.16	49.91	50.60	51.37	51.91
67	12.71	12.89	13.05	13.26	13.41
68	12.01	12.18	12.32	12.51	12.65
69	12.71	12.89	13.05	13.26	13.41
70	12.71	12.89	13.05	13.26	13.41
71	0.00	0.00	0.00	0.00	0.00
72	0.00	0.00	0.00	0.00	0.00

APPENDIX XII

Kaune Wire Coding Matrix

Wertheimer - Leeper Wiring Code for overhead electrical wires (Kaune 1990)				
	1	2	3	4
Wiring structure	VHCC	OHCC	OLCC	VLCC Endpole
1. Transmission line 2. Thick 3-phase primary 3. ≥ 6 primary phase wires (same pole)	≤ 15.2 m (50 ft)	≤ 39.6m (130 ft)		≥ 39.6m (130 ft)
1. Thin 3-phase primary	≤ 7.6m (25 ft)	≤ 15.2 m (50 ft)	≤ 39.6m (130 ft)	≥ 39.6m (130 ft)
1. First-span secondary		≤ 15.2 m (50 ft)	≤ 39.6m (130 ft)	≥ 39.6m (130 ft)
1. Second span (not endpole)			≤ 39.6m (130 ft)	≥ 39.6m (130 ft)

From William T. Kaune 1990

APPENDIX XIII

Industry Categories Considered "High Risk"

APPENDIX XIV

Occupation Categories Considered "High Risk"

Industrial Categories Considered "High Risk" Industries for Industry Category Analysis (Cont.)

TRANSPORTATION, COMMUNICATIONS, AND OTHER PUBLIC UTILITIES

Transportation
- Railroads
- Bus service and urban transit
- Taxicab service
- Trucking service
- Warehousing and storage
- U.S. Postal Service
- Water transportation
- Air transportation
- Pipe lines, except natural gas
- Services incidental to transportation

Communications
- Radio and television broadcasting
- Telephone (wire and radio)
- Telegraph and miscellaneous communication services

Utilties and sanitarty services
- Electric light and power
- Gas and steam supply systems
- Electric and gas, and other combinations
- Water supply and irrigation
- Sanitary services
- Not specified utilities

RETAIL TRADE

Gasoline service stations
Fuel and ice dealers

BUSINESS AND REPAIR SERVICES

Automotive repair shops
Electrical repair shops
Miscellaneous repair services

PERSONAL SERVICES

Laundry, cleaning, and garment services
Beauty shops
Barber shops

Industrial Categories Considered "High Risk" Industries for Industry Category Analysis (Cont.)

MANUFACTURING--Con.
Durable Goods--Con.

Metal industries--Con.
- Metal forgings and stampings
- Ordnance
- Miscellaneous fabricated metal products
- Not specified metal industries

Machinery, except electrical
- Engines and turbines
- Farm machinery and equipment
- Construction and material handling machines
- Metalworking machinery
- Office and accounting machines
- Electronic computing equipment
- Machinery, except electrical, n.e.c.
- Not specified machinery

Electrical machinery, equipment, and supplies
- Household appliances
- Radio, TV, and communication equipment
- Electrical machinery, equipemtn, and supplies, n.e.c.
- Not specified electrical machinery, equipment, and supplies

Transportation equipment
- Motor vehicless and motor vehicle equipment
- Aircraft and parts
- Ship and boat building and repairing
- Railroad locomotives and equipment
- Guided missiles, space vehicles, and parts
- Cycles and miscellaneous transportation equipment

Professional and photographic equipment, and watches
- Scientific and controlling instruments
- Optical and health services supplies
- Photographic equipment and supplies
- Watches, clocks, and clockwork operated devices
- Not specified professional equipment

Toys, amusement, and sporting goods

Miscellaneous manufacturing industries

Not specified manufacturing industries

Industrial Categories Considered "High Risk" Industries for Industry Category Analysis (Cont.)

AGRICULTURE, FORESTRY AND FISHERIES

MANUFACTURING--Con.
Nondurable Goods--Con.

Petroleum and coal products
- Petroleum refining
- Miscellaneous petroleum and coal products

Rubber and miscellaneous plastics products
- Tires and inner tubes
- Other rubber products, and plastic footwear and belting

Miscellaneous plastics products

Leather and leather products
- Leather tanning and finishing
- Footwear, except rubber and plastic
- Leather products, except footwear

Durable Goods

Lumber and wood products, except furniture
- Logging
- Sawmills, planing mills, and millwork
- Wood buildings, and mobile homes
- Miscellaneous wood products

Furniture and fixtures

Stone, clay, glass, and concrete products
- Glass and glass products
- Cement, concrete, gypsum, and plaster products
- Structural clay products
- Pottery and related products
- Miscellaneous nonmetallic mineral and stone products

Metal industries
- Blast furnaces, steelworks, rolling and finishing mills
- Iron and steel foundries
- Primary aluminum industries
- Other primary metal industries
- Cutlery, hand tools, and other hardware
- Fabricated structural metal products
- Screw machine products

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

MANAGERIAL AND PROFESSIONAL SPECIALTY OCCUPATIONS--Con
Technicians and Related Support Occupations--Con.

Health technologists and technicians--con.
Radiologic technicians
Licensed practical nurses
Health technologists and technicians, N.E.C.
Technologists and technicians, except health
Engineering and related technologists and technicians
Electrical and electronic technicians
Industrial engineering technicians
Science technicians
Biological technicians
Chemical technicians, Science Technicians, N.E.C.
Airplane pilots and navigators
Air traffic controllers
Broadcast equipment operators
Computer programmers

Administrative Support Occupations, Including Clerical

Coccumications equipment operators
Telephone operators
Telegraphers
Communications wquipment operators, N.E.C.

SERVICE OCCUPATIONS
Protective Service Occupations

Firefighting and fire prevention occupations
Fire inspection and fire prevention occupations
Firefighting occupations

FARMING, FORESTRY, AND FISHING OCCUPATIONS
Farm operators and managers
Farmers except horticultural

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis

MANAGERIAL AND PROFESSIONAL SPECIALTY OCCUPATIONS

Professional Specialty Occupations

Engineers, architects, and surveyors
- Engineers
  - Chemical engineers
  - Nuclear engineers
  - Electrical and electronic engineers
  - Industrial engineers
  - Mechanical engineers
  - Marine engineers and naval architects

Naturan scientists
- Physicists and astronomers
- Chemists, except biochemists
- Atmospheric and space scientists
- Geologists and geodesists
- Physical scientists, n.e.c.
- Agricultural and food scientists
- Biological and life scientists
- Forestry and conservation scientists
- Medical scientists

Health diagnosing practitioners, n.e.c.

Health assessment and treating occupations
- Registered nurses

Teachers, postsecondary
- Chemistry teachers
- Physics teachers
- Medical science teachers
- Health specialties teachers

Writers, artists, entertainers, and athletes
- Painters, sculptors, craft–artists, and artist printmakers

TECHNICAL, SALES, AND ADMINISTRATIVE SUPPORT OCCUPATIONS

Technicians and Related Support Occupations

Health technologists and technicians
- Clinical laboratory technologists and technicians

Industrial Categories Considered "High Risk" Industries for Industry Category Analysis (Cont.)

TRANSPORTATION, COMMUNICATIONS, AND OTHER PUBLIC UTILITIES

Transportation
- Railroads
- Bus service and urban transit
- Taxicab service
- Trucking service
- Warehousing and storage
- U.S. Postal Service
- Water transportation
- Air transportation
- Pipe lines, except natural gas
- Services incidental to transportation

Communications
- Radio and television broadcasting
- Telephone (wire and radio)
- Telegraph and miscellaneous communication services

Utilties and sanitarty services
- Electric light and power
- Gas and steam supply systems
- Electric and gas, and other combinations
- Water supply and irrigation
- Sanitary services
- Not specified utilities

RETAIL TRADE

Gasoline service stations
Fuel and ice dealers

BUSINESS AND REPAIR SERVICES

Automotive repair shops
Electrical repair shops
Miscellaneous repair services

PERSONAL SERVICES

Laundry, cleaning, and garment services
Beauty shops
Barber shops

Industrial Categories Considered "High Risk" Industries for Industry Category Analysis

AGRICULTURE, FORESTRY, AND FISHERIES

Agricultural production, crops
Agricultural services, except horticultural
Horticultural services

MINING

Metal mining
Nonmetallic mining and quarrying, except fuel

MANUFACTURING

Nondurable Goods

Tobacco manufactures
Textile mill products
- Knitting mills
- Dyeing and finishing textiles, except wool and knit goods
- Floor coverings, except hard surface
- Yarn, thread, and fabric mills
- Miscellaneous textile mill products

Apparel and finished textile products
- Apparel and accessories, except knit
- Miscellaneous fabricated textile products

Paper and allied products
- Pulp, paper, and paperboard mills
- Miscellaneous paper and pulp products
- Paperboard containers and boxes

Printing, publishing, and allied industries
- Newspaper publishing and printing
- Printing, publishing, and allied industries, except newspapers

Chemicals and allied products
- Plastics, synthetics, and resins
- Drugs
- Soaps and cosmetics
- Paints, varnishes, and related products
- Agricultural chemicals
- Industrial and miscellaneous chemicals

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

PRECISION PRODUCTION, CRAFT, AND REPAIR OCCUPATIONS--Cor

Mechanics and repairers--Con.
Electrical and electronic equipment repairers--Con.
Miscellaneous electrical and electronic equipment repairers
Heating, air conditioning, and refrigeration mechanics
Miscellaneous mechanics and repairers
Camera, watch, and musical instrument repairers
Locksmith and safe repairers
Office machine repairers
Mechanical controls and valve repairers
Millwrights
Specified mechanics and repairers, n.e.c.
Not specified mechanics and repairers
Precision production occupations
Supervisors, production occupations
Precision metal working occupations
Tool and die makers
Tool and die maker apprentices
Precision assemblers, metal
Machinists
Machinist apprentices
Boilermakers
Precision grinders, fitters, and tool sharpeners
Patternmakers and mold makers, metal lay-out workers
Precious stones and metals workers (jewelers)
Engravers, metal
Sheet metal workers
Sheet metal worker apprentices
Precision woodworking occupations
Patternmakers and model makers, wood
Cabinet makers and bench carpenters
Furniture and wood finishers
Miscellaneous precision woodworkers
Precision workers, assorted materials
Hand molders and shapers, except jewelers
Patternmakers, lay-out workers, and cutters

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

FARMING, FORESTRY, AND FISHING OCCUPATIONS--Con.

Farm operators and managers--Con.
- Horticultural specialty farms
- Managers, farms, except horticultural
- Managers, horticultural specialty farms

Other agricultural and related occupations
- Farm occupations, except managerial
  - Supervisors, farm workers
  - Farm workers
  - Nursery workers
- Related agricultural occupations
  - Supervisors, related agricultural occupations
  - Groundkeepers and gardeners, except farm

PRECISION PRODUCTION, CRAFT, AND REPAIR OCCUPATIONS

Mechanics and repairers
- Supervisors, mechanics and repairers
- Mechanics and repairers, except supervisors
  - Vehicle and mobile equipment mechanics and repairers
  - Automobile mechanics
  - Automobile mechanic apprentices
  - Bus, truck, and stationalry engine mechanics
  - Aircraft mechanics, except engine
  - Small engine repairers
  - Automobile body and related exposures
  - Farm equipment mechanics
  - Industrial machinery repairers
  - Machinery maintenance occupations

Electrical and electronic equipment repairers
- Electronic repairers, commumications and industrial equipment
- Data processing equipment repairers
- Household appliance power tool repairers
- Telephone line installers and repairers
- Telephone installers and repairers

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

PRECISION PRODUCTION, CRAFT, AND REPAIR OCCUPATIONS--Cor

Precision workers, assorted materials--Con.
Optical goods workers
Dental laboratory and medical appliance technicians
Bookbinders
Electrical and electronic equipment assemblers
Miscellanious precesion workers, n.e.c.
Precision inspectors, testers, and related workers
Inspectors, testers and graders
Adjusters and calibrators
Plant and system operators
Water and sewage treatment plant operators
Power plant operators
Stationary engineers
Miscellaneous plant and system operators

OPERATORS, FABRICATORS, AND LABORERS
Machine Operators, Assemblers, and Inspectors

Machine operators and lenders, except precision
Metalworking and plastic working machine operators
Lathe and turning machine set-up operators
Lathe and turning machine operators
Milling and planning machine operators
Puching and stamping press machine operators
Rolling machine operators
Drilling and boring machine operators
Grinding, abrading, buffing, and polishing machine operators
Forging machine operators
Numerical control machine operators
Miscellaneous metal, plastic, stone, and glass working machine operators
Fabricating machine operators, n.e.c.
Metal and plastic processing machine operators
Molding and casting machine operators
Metal plating machine operators
Heat treating equipment operators

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

OPERATORS, FABRICATORS, AND LABORERS--Con.
Machine Operators, Assemblers, and Inspectors--Con.

Machine Operators and lenders, except precision--Con.
Metal and plastic processing machine operatoes--Con
Miscellaneous metal and p;astic processing machine operators
Sawing machine operators
Shaping and joining machine operators
Nailing and tacking machine operators
Miscellaneous woodworking machine operators
Printing machine operators
Printing machine operators
Photoengravers and lithographers
Typesetters and compositors
Miscellaneous printing machine operators
Textile, apparel, and furnishings machine operators
Winding and twisting machine operators
Knitting, looping, taping, and weaving machine operators
Textile cutting machine operators
Textile sewing machine operators
Shoe machine operators
Pressing machine operators
Laundering and dry cleaning machine operators
Miscellaneous textile machine operators
Machine operators, assorted materials
Cementing and gluing machine operators
Packaging and filling machine operators
Mixing and blending machine operators
Separating, filtering, and clarifying machine operators
Compressing and compacting machine operators
Painting and paint spraying machine operators
Roasting and baking machine operators, except food
Washing, cleaning, and pickling machine operators
Folding machine operators
Furnace, kiln, and oven operators, except food
Crushing and grinding machine operators
Slicing and cutting machine operators
Motion picture projectionists

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

OPERATORS, FABRICATORS, AND LABORERS--Con.
Machine Operators, Assemblers, and Inspectors--Con.

Machine operators and tenders, except precision--Con.
Machine operators, assorted materials
Photographic precess machine operators
Miscellaneous machine operators, n.e.c.
Machine operators, not specified
Fabricators, assemblers, and hand working occupations
Welders and cutters
Solderers and brazers
Assemblers
Handcutting and trimming occupations
Hand molding, casting, and forming occupations
Hand painting, coating, and decorating occupations
Hand engraving and printing occupations
Hand grinding and polishing occupations
Miscellaneous hand working occupations
Production inspectors, testers, samplers, and weighers
Production inspectors, checkers, and examiners
Production testers
Production samplers and weighers
Graders and sorters, except agricultural

Transportation and Material Moving Occupations

Motor vehicle operators
Supervisors, motor vehicle operators
Truck drivers, light
Driver-sales workers
Bus drivers
Taxicab drivers and chauffeurs
Parking lot attendants
Motor transportation occupations, n.e.c.
Transportation occupations, except motor vehicles
Rail trasnportation occupations
Railroad conductors and yardmasters
Locomotive operating occupations
Railroad brake, signal, ans swich operators
Rail vehicle operators, n.e.c.

Industrial Categories Considered "High Risk" Occupations for Occupation Category Analysis (Cont.)

OPERATORS, FABRICATORS, AND LABORERS--Con.
Transportation and Material Moving Occupations--Con.

Transportation occupations, except motor vehicles--Con.
Water trasnportation occupations
Ship captains and mates, except fishing boats
Sailors and deckhands
Marine engineers
Material moving equipment operators
Supervisors, material moving equipment operators
Operating engineers
Longshore equipment operators
Hoist and winch operators
Crane and tower operators
Excavating and loading machine operators
Grader, dozer, ans scraper operators
Miscellaneous material moving equipment operators

Handlers, Equipment Cleaners, Helpers, and Laborers

Supervisors, handlers, equipment cleaners, and laborers, n.e.c.
Helpers, mechanics and repairers
Production helpers
Freight, stock, and material handlers
Machine feeders and offbearers
Garage and service station related occupations
Laborers, except construction

APPENDIX XV

Frequency of Illnesses Identified Among Subjects

Appendix XV: Frequency of illnesses identified among study subjects which occurred prior to the date of case diagnosis.

Parent	Cases	Controls	Odds Ratio * O.R. (95% C.I)
Infectious Mononucleosis	–	–	
Allergies	–	–	
Asthma	–	–	
Chicken Pox	11	32	0.15 (0.03, 10.68)
Rubella (German Measles)	–	–	
Rubcola (measles)	–	–	
Mumps	2	6	–
Shigella Infection	–	–	
Salmonella Infection	1	1	–
Aplastic Anemia	–	–	
Cytomegalovirus	–	–	
Urinary Tract Infection	3	4	
Cancer	19	0	

* Odds ratios calculated for illnesses in which the total reported incidence for cases plus controls is greater than or equal to ten. Odds ratios as estimates of relative risk calculated using a conditional logistic regression model.

APPENDIX XVI

Birth Defects Considered During Interview

APPENDIX Birth Defects Considered During Interview Among Cases and Control

Downs Syndrome

Bloom's Syndrome

Klinefelter's Syndrome

X-Linked Agammaglobulinemia

Ataxia Telangiectasia

Wiskott-Aldrich Syndrome

APPENDIX XVII

Illnesses and Medications Considered During Interview

Illnesses and medications considered during interview of study subject mothers. No significant relationships were identified when the below list was considered.

ILLNESS	MEDICATIONS
Measles	Amphetamines
Rubella	Chloramphencol
Chickenpox	Chlorpromazine
Influenza	Griseofulvin
Hepatitis	Oxyphenbutazone
Genital Herpes	Phenylbutazone
Fever for more than 3 consecutive days	Propylthiouracil
Vaginal infections	Tolbutamide
Mononucleosis	
Vaginal or Uterine Bleeding	
High Blood Pressure	
Pre-eclampsia or toxemia	
Protein in urine	
Urinary tract infection	
Nausea or vomiting for over 3 months	
Cytomegalovirus infection	
Aplastic anemia	

APPENDIX XVIII

Medical Diagnostic Procedures Considered During Interview

APPENDIX

Medical diagnostic procedures considered during interview of study subject mothers. None of these procedures were reported by mother in the 3 months before pregnancy or during pregnancy.

Amniocentesis

Ultrasound

Upper G.I. Series

Gall Bladder X-Ray

Barium Enema

IVP or Cystogram

Lower Abdominal X-Ray

Back X-Ray

X-Ray of the fetus

Radiation therapy

Abdominal X-Ray

APPENDIX XIX

Public Comments and Responses

Cambridge Environmental Inc
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617-225-0810 FAX 617-225-0813 E-mail camenv58@aol.com

July 8, 1996

Kevin Costas, Ph.D.
Bureau of Environmental Health Assessment
Massachusetts Department of Public Health
250 Washington Street
Boston, MA 02108

Dear Dr. Costas:

We write to comment on the Draft Final Report of the *Woburn Childhood Leukemia Follow-up Study* by the Bureau of Environmental Health Assessment of the Massachusetts Department of Public Health (DPH), released for public comment on May 7, 1996. We believe that studying childhood leukemia in Woburn is difficult, and we respect and commend you and your colleagues for undertaking this complex task. We hope that the criticisms offered below will be of use as you finalize your analyses.

W.R. Grace & Co. sponsored us in preparing these comments. The opinions, however, are ours and we take responsibility for any errors or omissions.

Summary

The *Woburn Childhood Leukemia Follow-up Study* describes a case-control study of the association between exposure to water delivered from wells G and H in East Woburn and the incidence of childhood leukemia. Exposure to water from wells G & H was ascertained during four "etiologic" periods: (1) from two years before conception to case diagnosis, (2) during the two years before case conception, (3) during the mother's pregnancy with the case, and (4) from birth of the case to case diagnosis. The study found that consumption of water from wells G and H as a child was not associated with subsequent development of leukemia, although a mother's consumption of such water while pregnant was a predictor of childhood leukemia incidence. The confidence interval about even this association was very wide, as expected given the small number of cases and controls available for analysis. No confidence interval about a measure of the association between leukemia and consumption of water from wells G and H excluded the null.

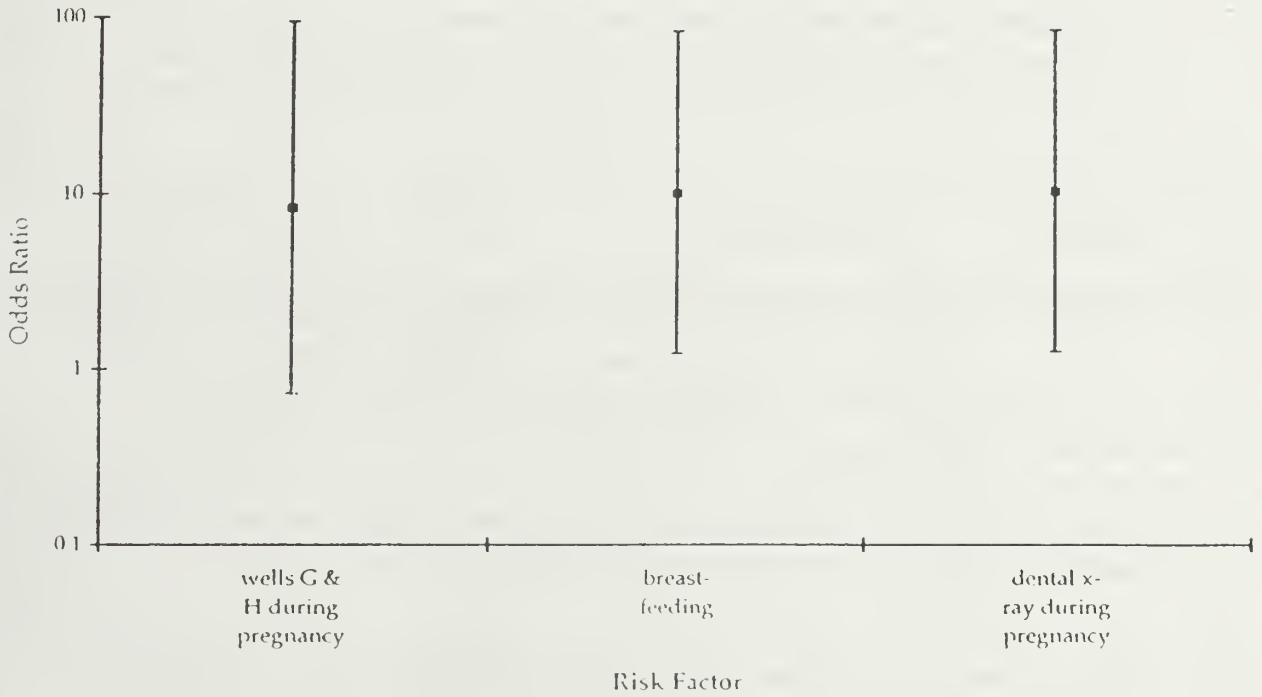
The authors examined a wide range of exposures and personal characteristics as suspected risk factors. Several key associations reported in the study are in the opposite direction of those typically found in other studies. For example, the study

suggests that maternal smoking during pregnancy, living in homes near electric power lines, and being born to an older mother are protective against childhood leukemia, and that breast-feeding is a strong risk factor for this disease. These anomalous results raise suspicion that the study suffered from underlying methodologic shortcomings that, when combined with its small sample size, yielded a series of imprecise and unreliable associations. Compounding the problem, the interpretation of associations observed in the study is inconsistent. For example, regarding the adjusted odds-ratio between exposure to wells G and H during pregnancy and incidence of childhood leukemia (OR = 8.33; 95% CI 0.73, 94.67), the DPH wrote (page 92):

> Strikingly, however, the adjusted odds ratio for maternal exposure during pregnancy suggests that the risk of developing childhood leukemia was greater for those children whose mothers consumed water from Wells G and H during pregnancy than for those whose mothers did not . . . . A mother's consumption of contaminated public water while pregnant with the case was the strongest predictor of childhood leukemia incidence.

The relative risk of childhood leukemia associated with breast-feeding during pregnancy, independent of wells G and H exposure, was 10.17 (95% CI 1.22, 84.5). The relative risk of childhood leukemia associated with dental x-ray during pregnancy was 10.49 (95% CI 1.26, 87.1). Both are stronger predictors of childhood leukemia incidence in this study than is exposure to wells G and H. However, the first association is characterized in the report (page 99) as "uncertain;" the relationship between breast-feeding and childhood leukemia is described as "unclear." The second association is characterized as "unstable."

Odds Ratios and Confidence Intervals about Three Largest Risk Factors
in the Woburn Childhood Leukemia Follow-up Study



The accompanying figure illustrates that the odds ratios reported in the study for these three risk factors are essentially equivalent. It follows that each risk factor should either receive equal and balanced treatment in the text of the report, or an explanation for a preference for one over the others should be offered.

We offer the following major comments, the first of which we expound upon in the first attachment (Technical Support). We have also attached a series of more minor points as a second attachment (Minor Comments) and a reference list as a third attachment (Literature Cited).

Major Points

1. Selection and control of confounders

A study is said to be confounded when the rates of disease due to background causes (*i.e.*, causes of disease other than the cause of interest) in the exposed and unexposed groups are unequal. Thus, confounders are both related to (that is,

cause) the disease under study and imbalanced between the exposed and unexposed groups. It is easy to see, for example, how smoking would confound the relationship between alcohol ingestion and oral cancer. Cigarette smoking is a cause of oral cancer and people who ingest higher amounts of alcohol (the exposed) are more likely to smoke than those who ingest lower amounts of alcohol (the unexposed).

It is not so easy to see, however, how the long list of variables controlled for in the DPH study could all confound the relationship between the incidence of childhood leukemia and exposure to wells G and H. Exposure to wells G and H is likely to be evenly balanced in the study population: one does not expect there to be behavioral, physical, or social attributes that both cause childhood leukemia and are associated with greater or lesser likelihood of being exposed to water from wells G and H. Nonetheless, the DPH controlled for confounding by age, sex, and resident in Woburn at the time of case diagnosis by matching.[1] DPH further controlled for confounding by socioeconomic status, maternal smoking during pregnancy, maternal age at birth of child, and maternal alcohol consumption during pregnancy using a composite covariate and multivariate conditional logistic regression.

The effect of controlling for variables that are not significant confounders is ordinarily only to reduce the precision of the estimate of effect. When control of variables strongly suspected *a priori* not to confound the association of interest does, in fact, substantially change the measure of association, that change may signal a bias in selection of the controls rather than successful control of a confounder (Kleinbaum *et al.*, 1982). Such a circumstance appears to have arisen in the DPH study. Two observations support this line of reasoning.

First, despite the strong prior belief that few variables would be strongly associated with exposure to wells G and H, control of these variables as if they were confounders dramatically affected the measure of association. The following table shows the crude measure of association between exposure to wells G and H during each of the etiologic periods and incidence of childhood leukemia, the measure

---

[1] As one peer reviewer pointed out, matching does not actually control for confounding in a case-control study design. Rather, it introduces a selection bias which is then controlled by matched analysis, such as conditional logistic regression. The purpose of matching in a case-control study is to allow a more efficient analysis of the association while controlling for confounders.

adjusted for the matching factors, and the measure adjusted for the matching factors and the composite covariate.

Table [1] Estimated relative risk of childhood leukemia among those ever exposed to wells G and H compared with those never exposed

Etiologic Period	Crude[a]	Adjusted for Matching[b]	Adjusted for Matching and Composite Covariate[c]
full	2.89 (0.71, 11.78)	1.99 (0.52, 7.71) -48%	2.39 (0.54, 10.59) 40%
2 yrs < conception	1.52 (0.48, 4.75)	1.77 (0.39, 8.00) 48%	2.61 (0.47, 14.37) 109%
during pregnancy	2.31 (0.75, 7.2)	5.70 (0.67, 48.25) 259%	8.33 (0.73, 94.67) 56%
birth to diagnosis	1.31 (0.42, 4.07)	1.36 (0.00, 5.38) 16%	1.18 (0.28, 5.05) -50%

[a] odds ratio with 95% confidence interval in parentheses
[b] odds ratio with 95% confidence interval in parentheses, % change in excess relative risk compared with crude
[c] odds ratio with 95% confidence interval in parentheses, % change in excess relative risk compared to adjusted for matching

As shown in the table, controlling for the matched factors depresses the odds ratio compared with the crude for the full etiologic period (from two years before conception to case diagnosis) yet increases the odds ratio for each of the three subperiods. We know of no explanation for why the odds ratio would move downward after adjusting for the matched factors (age, sex, and resident in Woburn at diagnosis) over the entire etiologic period but move upward after adjusting for

the matched factors within each subperiod. Similarly, controlling for confounding by the composite covariate moves the odds ratio upward for three of the etiologic periods and downward for the fourth. The implication is that the direction of the selection bias introduced by matching and the confounding introduced by the covariates depends on the definition of exposure. The dependence is not the same for the matched factors as it is for the covariates. Given the nature of the matched factors and covariates, it is difficult to imagine an underlying mechanism that would explain these inconsistencies.

Second, one must consider whether the size and direction of the confounding influence of the covariates is consistent with expectation. Recall that the composite covariate is meant to permit control for confounding by socioeconomic status, maternal smoking during pregnancy, maternal age at birth of child, and maternal alcohol consumption during pregnancy. We expect *a priori* the confounding influence of these variables to be low, since none should be strongly related to the ever/never dichotomization of exposure to wells G and H.[2] Nonetheless, Table 1 shows that control for these confounders had a large impact on the size of the odds ratio.

In addition, of the four confounding variables included in the composite covariate: (i) the univariate odds-ratio for socioeconomic status was not reported; (ii) the univariate odds-ratios for maternal age at pregnancy with the index child and the odds-ratio for maternal smoking during pregnancy suggest increasing risk in the direction opposite of what is usually measured; and (iii) the univariate odds-ratio for maternal ingestion of alcohol during pregnancy is in the direction and of the size expected. The associations with three other risk factors are inconsistent with the reported literature. Dental x-ray during pregnancy and breast-feeding are both important risk factors for childhood leukemia in this study, but they are only minor (for x-irradiation) or null (for breast feeding) risk factors in the literature. Exposure to electromagnetic fields is a positive risk factor in the literature, but apparently

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[2] We further expect these confounders to be closely correlated. For example, control of confounding by maternal smoking during pregnancy likely goes a long way towards controlling for maternal alcohol consumption during pregnancy. Similarly, control of confounding by socioeconomic status likely controls, at least in part, for confounding by maternal age at birth of child.

protective in this study. These inconsistencies require explanation in the study. We suggest that they may be indicators of a bias in control selection.

Most important, control for confounders such as maternal age and smoking during pregnancy — which yielded an association in the opposite direction of what was expected — will inflate the adjusted estimate of effect when proper control should reduce the measure of effect. The odds-ratios adjusted for both the matched factors and the composite covariate overestimate the risk associated with exposure to wells G and H because of this bias. The odds-ratios adjusted for the matched factors alone also overestimate the risk; they should be adjusted downward to take account of the real direction of the confounding by the covariates with associations in this study that are opposite of the expected direction.

2. Inconsistency with Lagakos *et al.* (1986)

The results of this study are not consistent with a previous study of the effect of exposure to wells G and H and the incidence of childhood leukemia (Lagakos *et al.*, 1986).[3] Lagakos *et al.* (1986) found an apparent association between exposure to wells G and H from birth to case diagnosis, whereas this study found no such association. Lagakos *et al.* (1986) wrote:

> Using either the cumulative ($P = .03$, $\hat{\alpha} = .33$) or none versus some ($P = .02$, $\hat{\alpha} = 1.11$) exposure metrics, there is a positive association between G and H exposure and the incidence rate of childhood leukemia.

Lagakos *et al.* (1986) determined for each child an exposure "history," consisting of his or her set of annual exposure scores, beginning from the first year of Woburn residency. This exposure definition would therefore be comparable to the DPH study's etiologic period from birth to case diagnosis. For that etiologic period, the DPH measured an odds ratio associating the incidence of childhood leukemia with

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[3] Inconsistency between the results of the current study and the results of the Lagakos *et al.* (1986) study are not necessarily unexpected. As outlined here in section 3, and as noted by other reviewers (MacMahon, 1986; Prentice, 1986; Rogan, 1986; Swan and Robins, 1986; and Whittemore, 1986), the Lagakos *et al.* (1986) study had "several major drawbacks" (ATSDR, 1993) that rendered the results unreliable.

exposure to wells G and H of 1.36 (95% CI 0.00, 5.38) adjusting for the matched factors and 1.18 (95% CI 0.28, 5.50) adjusting for the matched factors and the composite covariate. The DPH says "no such relationship" (*i.e.*, elevation of the odds ratio) "was identified in the time period between birth and diagnosis." The results of the Lagakos study and the DPH study are therefore inconsistent, although both studied essentially the same set of cases and both ascertained exposure to wells G and H during the same etiologic period by very similar methods.

The second paragraph on page 105 of the study reads:

> Although detail regarding exposure during specific times within the subject's lifetime are not discussed (in Lagakos *et al.*, 1986), the general conclusions are similar to those we have drawn specific to the Murphy Model exposure metric and the current study's exposure values during pregnancy. Lagakos *et al.* found an association in their ever/never exposure analysis between exposure and childhood leukemia as we did.

As explained above, the results from comparable etiologic periods are not, in fact, "similar."

3. A case of leukemia secondary to chemotherapy to treat lymphosarcoma should be deleted

Lagakos *et al.* (1986) should have excluded one case of childhood leukemia because the child's disease did not satisfy the case definition. In previous work reviewing the Lagakos *et al.* (1986) study, one of us (CP) found that one case (number 4 in Lagakos *et al.*, 1986) was diagnosed initially with lymphosarcoma and then developed leukemia subsequent to chemotherapy. Dr. Alvin Young, at that time Director of the SEER program, confirmed that childhood leukemia subsequent to lymphosarcoma should not be considered an incident case of childhood leukemia. This case of lymphosarcoma was highly influential to the results of Lagakos *et al.* (1986) because the authors in advisedly compared the sum of observed and expected exposure scores for the cases. This method, which is equivalent to comparing mean exposure scores between cases and controls, is unreliable (Rothman, 1986). One reason is that it is highly susceptible to undue influence by extreme values. This exposure score for the lymphosarcoma case was one such value, being the second

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highest in the entire analysis, an analysis in which only nine cases had above-zero exposure scores. We cannot tell what the influence of the lymphosarcoma case is in the present study, or even be certain that it is included. That is because this report, unlike all previous reports on childhood leukemia in Woburn, did not provide a table giving information on each case. Although this is the most obvious and important error we found in reviewing the Lagakos *et al.* (1986) work (which work provided the starting point for case ascertainment in the present study), we found other possible errors and discrepancies that also might have been carried through to this study, not only in case-finding but in residence locations and other aspects.

4. The rationale for case eligibility dates requires explanation

The DPH defined a case as a child who 1) was diagnosed with leukemia prior to his or her 19th birthday, 2) was diagnosed on or after January 1, 1969, and 3) was a resident of Woburn at the time of his or her diagnosis. This definition omits a crucial aspect of the design of any epidemiologic study: the date on which the followup or case ascertainment period ended.

A summary of an MDPH 1981 report contained in the discussion section suggests that childhood leukemia mortality was not elevated in Woburn during the period between 1949 and 1958 but began to rise between 1959 and 1963. No unusual geographic distribution of childhood leukemia mortality occurred from 1949 to 1968. Well G began operating in October 1964. To measure the mortality rate between 1949 and 1968, the DPH must have had access to reports of childhood leukemia of some sort. It seems that with some effort the cases arising before 1969 could have been discovered and included in this study. The DPH should explain its choice of the January 1, 1969 case eligibility date.

The DPH also effectively closed case detection in August 1989, although the case definition includes no end-eligibility date. The first paragraph of the Introduction section says that new cases of childhood leukemia arose in 1994. The first paragraph of the Discussion section includes the sentence, "Two new cases were diagnosed, one each in February and March of 1994 among Woburn residents." The second paragraph of the Discussion section echoes this information. These two cases meet the written case definition. The DPH should explain its choice of the August 1989 end-eligibility date and should explain why the two 1994 cases were excluded from the analysis.

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5. The study was very small and, for exposure to wells G and H *in utero*, it was made effectively much smaller by the date-of-birth and risk set matching.

The study results rely on information provided for only twenty-one cases, two of which did not participate, leaving nineteen cases and thirty-seven matched controls. Such a study is assured from the start to provide only imprecise measures of the association between any exposure and disease.

The matching procedure, the purpose of which is to improve the precision of case-control analyses, in this study had the opposite effect. Every case who was born after October 1964 and every case whose mother's last menstrual period was after May 1979 was of necessity unexposed and, by virtue of the matching, was of necessity matched to an unexposed control. These concordant case-control sets do not contribute to analyses that control for the matched factors. Thus, in all matched analyses, the study reduces to a study of children who were *in utero* sometime during the interval October 1964 to May 1979. Based on a prior analysis of Lagakos *et al.* (1986), we estimate that, not counting the case of lymphosarcoma that should have been excluded from the study entirely, the matching had the effect of excluding eight, or 44%, of the eighteen available cases (twenty-one less two non-participants, less one ineligible case of lymphosarcoma).

Additional details and comments are presented in the attachments to this letter.

Thank you for your consideration of these comments.

Sincerely,

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Attachment 1: Technical Support for Major Point 1

Univariate analysis of the association between many of the confounding variables and the incidence of childhood leukemia yielded an association opposite to the association most often reported in the literature.

The study does not report its method of measuring parents' socioeconomic status, nor does it report a univariate analysis of the association between socioeconomic status and incidence of childhood leukemia. Socioeconomic status has been positively related to the incidence of childhood leukemia and may be related to biases in control selection (Poole and Trichopolous, 1991).

The study reports that five control mothers and no case mothers smoked during pregnancy with the control or case child. The odds ratio relating smoking during pregnancy to the incidence of childhood leukemia in this study therefore equals zero. The p-value associated with the observation is p = 0.12. The literature reports that association between maternal smoking and/or passive smoking and incidence of childhood leukemia is null or slightly elevated (Tredaniel *et al.*, 1994). The literature review provided by the DPH centers on smoking as a direct cause of leukemia in adults, not on passive smoking or *in utero* exposure to cigarette smoke inhaled by the mother as a risk factor for childhood leukemia.

The study reports an odds-ratio for "increasing maternal age"[4] of 0.55 (95% CI 0.14, 2.07), so that children born to younger mothers were at higher risk for childhood leukemia than children born to older mothers. This result is opposite to the direction usually reported in the literature. For example, Shiu *et al.* (1995) reported that mothers of children diagnosed with acute leukemia had a mean age at birth of the case of 28.4 yrs (SD = 2.7 years) whereas mothers of control children matched on sex and year of birth had mean age of 27.8 years (SD = 2.4 years) (t-test for homogeneity of means yields p = 0.037). MacMahon and Newille (1962) observed a positive association between increasing maternal age and incidence of acute lymphoblastic leukemia

[4] It would be helpful for the DPH to include a description of the parameterization of maternal age. Was it dichotomized, treated as a continuous variable, or treated in some other way?

The study reports a matched odds-ratio of 1.5 (95% CI 0.54, 4.20) for maternal ingestion of alcohol during pregnancy. The expected correlation between ingestion of alcohol during pregnancy and smoking during pregnancy was not observed.

Several other associations reported in the study were inconsistent with the values most often reported in the literature. The study reports children who were breast fed were at 10.17-fold (95% CI 1.22, 84.5) greater risk of childhood leukemia than children who were not breast fed, independent of the influence of other risk factors. Shiu *et al.* (1995) reported an odds ratio of 1.14 (95% CI 0.7, 1.9) for risk of acute childhood leukemia among ever breast-fed versus never breast-fed children. Magnani *et al.* (1988) reported an odds ratio of 1.1 (95% CI 0.7, 1.6) for risk of acute lymphoblastic leukemia among children breast-fed zero to six months versus children never breast-fed and an odds ratio of 1.1 (95% CI 0.6, 2.3) for risk of acute lymphoblastic leukemia among children breast-fed seven or more months versus children never breast-fed. Magnani *et al.* (1988) also reported an odds ratio of 1.2 (95% CI 0.5, 3.0) for risk of acute non-lymphoblastic leukemia among children breast-fed zero to six months versus children never breast-fed and an odds ratio of 0.5 (95% CI 0.1, 4.3) for risk of acute non-lymphoblastic leukemia among children breast-fed seven or more months versus children never breast-fed. Similarly, van Duijn *et al.* (1988) report an odds ratio of 1.2 (95% CI 0.8, 1.7) for risk of acute lymphoblastic leukemia among children breast-fed zero to six months versus children never breast-fed and an odds ratio of 0.8 (95% CI 0.5, 1.4) for risk of acute lymphoblastic leukemia among children breast-fed seven or more months versus children never breast-fed. Schwartzbaum *et al.* (1991) report an odds ratio of 1.0 for risk of acute lymphoblastic leukemia and 0.8 for acute non-lymphoblastic leukemia among children breast-fed eight months or more versus children breast-fed for less than two months. Thus, the literature indicates a null to protective effect for children breast-fed for shorter duration and more strongly indicates a protective effect for children breast-fed for longer durations. The odds ratio reported by the DPH is clearly inconsistent with the body of literature on the relationship between breast-feeding and childhood leukemia.

The study reports that children whose mothers had dental x-rays during pregnancy were at 10.5-fold higher risk (95% CI 1.3, 87.1) for childhood leukemia than children whose mothers did not have dental x-rays during pregnancy. Graham *et al.* (1966) present a summary of five measurements of relative risk of childhood leukemia associated with abdominal x-ray during pregnancy. The measurements of

relative risk range from 1.27 to 1.89. Graham *et al.*'s (1966) study measured a relative risk of 1.7 (95% CI 1.3, 2.2) for x-ray to all sites during pregnancy. None of these studies measured the relative risk associated with dental x-ray *per se*, although one would expect the total x-ray dose received by x-irradiation of the abdomen to be greater than from x-ray of only the mouth.

Finally, the study reported that children living in high-current code homes were at half the risk (OR=0.53; 95% CI 0.13, 2.08) of childhood leukemia when compared with children living in low current code homes. Most of the published studies report a positive association between exposure to electromagnetic fields and incidence of childhood leukemia (Wertheimer and Leeper, 1979; Savitz *et al.*, 1988; London *et al.*, 1991; Feychting and Ahlbom, 1993; Verksalo *et al.*, 1993; and Olsen *et al.*, 1993).

Attachment 2: Minor comments

1. Page 2 of the Executive Summary says that controls were matched to cases based on "age, sex, race, and dates of residence in Woburn." Page 30 of the report says that controls were matched to cases on "race, sex, and age ± 3 months from the case's date of birth. Controls must have been Woburn residents at the time of the diagnosis of the matched case." The description of matching offered in the Executive Summary is ambiguous. Controls were matched to cases on date-of-birth, not just on age. Furthermore, as written in the Executive Summary, the description of matching on Woburn residence could imply that controls had to have the same residential history as the cases, when in fact they needed to be a resident in Woburn only at the time of the case's diagnosis.[5]

2. The definition of controls offered on page 27 of the report does not correspond to the modern definition. The report states "Controls are a group of individuals in whom the disease is absent and who have been selected for comparison based on specific matching criteria." Under the modern definition, controls are a sample of the population that produced the cases. They do not have to be disease-free and they should not be thought of as the "comparison" group: all comparisons are between exposed and unexposed subjects.

3. The explanation of the purpose of matching in case-control studies on page 27 is not correct. Matching in case-control studies does not balance disease risks within exposure groups. Rather, matching allows for more efficient control of confounding in the analysis by assuring a more even balance of subjects within strata of the matched factors than would be obtained by unmatched

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[5] Note also that the second paragraph of page 51 indicates that controls were matched to cases on duration of Woburn residency, not residency in Woburn at the time of case diagnosis. The paragraph says "Residential information for each case was comparable by study design to that of its two matched controls. This means that each matched triplet (the case and its two controls) had Wells G and H water exposure values based on the same length of residence in Woburn."

sampling. A peer reviewer made this point also and it has not been fully corrected in the text.

4. Since controls were all selected from public school records, readers might be concerned that cases attending private school would not have arisen from the population sampled to obtain controls. A lack of correspondence between cases and the base that produced the cases is indeed cause for concern in case-control studies. We have learned (DPH, personal communication, 1996) that all of the cases who attended school did, in fact, attend public school in Woburn. This observation should be included in the text of the report.[6]

5. The report states on page 60 that "Variables where positive responses occurred at a total frequency (cases and controls) of at least 10 and whose relationship with both leukemia and water exposure yielded an odds ratio greater than or equal to 1.5 were entered into the multivariate model." This is an unconventional method of detecting confounders. First, we know of no basis for requiring at least ten positive responses among cases and controls. Second, the association between the confounder and disease should be measured only among the unexposed. The association between the confounder and wells G and H exposure should be measured only among the controls (Kleinbaum *et al.*, 1982). Third, the more conventional technique for detecting confounders is to choose those variables for which the adjusted (for the potential confounder) measure of effect[7] differs appreciably from the unadjusted measure of effect and for which there is no substantial evidence of

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[6] We note that the DPH addressed this issue on page 12 of its response to peer reviewer comments, but did not mention in that response that all cases who reached school age attended public school.

[7] Preferably the standardized morbidity ratio.

effect modification by the potential confounder.[8] The DPH should explain why it chose its method for detecting confounders.

6. The report states on page 61 that the p-values "give the probability that the null hypothesis (of no relationship) is true given the data." This is an incorrect definition of the p-value. The "truth" of the null hypothesis must be measured by the validity of the study methods as well as its precision. The p-value can measure only the precision of an estimate of effect. We suggest a definition such as "the p-value is the probability of observing the study's results, or results more extreme, assuming that the null hypothesis is true. The p-value cannot incorporate a measurement of the validity of the study's methods."

7. Similarly, the report states on page 66 that "The narrower the confidence interval, the more likely the odds ratio truly represents the amount of risk among cases relative to controls." This definition has two shortcomings. First, the confidence interval provides no information on the "truth" of the odds ratio: it is only a measure of a study's precision, not of its validity. Second, the comparison in a case-control study, as in all etiologic epidemiology studies, is between exposed and unexposed subjects, not between cases and controls. We suggest a definition such as "The width of the confidence interval provides a measure of the precision of the association; it cannot incorporate a measurement of the validity of the study's methods."

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[8] The report says (p. 60) that "likelihood statistics were sometimes employed to evaluate the importance of interaction terms." These interaction terms were likely the cross-product between the main effect and the covariate, a measure of interaction on the multiplicative scale. The interaction of biologic relevance is interaction on an additive scale (Rothman, 1986). In any event, the statistical significance of the interaction term as measured by likelihood statistics is a poor measure of effect modification. We suggest that the DPH investigate interaction by calculating the odds ratios within strata of the potential effect modifiers and looking for substantial deviation from additivity of the rates as described by Rothman (1986).

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8. The authors write on page 95 that control of confounding using the Tukey method composite covariate reduced the width of the confidence interval by half compared with the multivariate model entering each covariate individually. The variance of the odds ratio, and so the width of the confidence intervals, should be measured on the log-scale. Transforming the values shown in Table 12 of the text, we obtain a ln-odds ratio adjusted using the Tukey composite covariate of 2.12, a ln-LCL95 of -0.32 and a ln-UCL95 of 4.55, yielding a confidence-interval width of 4.87. We obtain a ln-odds ratio adjusted using the individual covariates of 2.58, a ln-LCL95 of -0.16 and a ln-UCL95 of 5.32, yielding a confidence-interval width of 5.48. On the log scale, then, the confidence interval about the odds-ratio adjusted by entering each variable individually into the multivariate model is only 1.13-fold wider than the confidence interval about the odds-ratio adjusted by using the Tukey covariate. The confidence interval is not twofold wider. Of course, the Tukey method does not account for the standard error of the score-variable assigned to each subject, so one would expect at least the appearance of greater precision using this method.

9. We have enclosed a copy of an unpublished study by the Arizona Department of Health Services (Flood *et al.*, 1990) that the DPH may wish to include in its discussion of the carcinogenicity of trichloroethylene. Flood *et al.* (1990) studied the incidence of childhood leukemia in Maricopa County from 1965 to 1986. A cluster of childhood leukemia cases was observed in the area, just as in Woburn. One of the *a priori* hypothesis of the study was that the cluster was related to ingestion of trichloroethylene in contaminated water. The study "failed to demonstrate an association with . . . proximity to wells which pumped TCE-contaminated water."

10 Table 22 of the study indicates that the DPH gathered information on maternal consumption of tapwater during the pregnancy with the case or control child. Yet, this information was not apparently included in the exposure metric. The DPH should provide crude and adjusted estimates of effect for the etiologic period "during pregnancy" with exposure defined as it currently has been defined but also requiring an indication of consumption of household tapwater during pregnancy in response to question 23 of the survey.

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July 15, 1997

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Dear Sirs:

This document is in response to your letter providing major and minor comments concerning the Draft Final Report of The Woburn Childhood Leukemia Follow-up Study. Your major comments are addressed herein. Minor comments have been reflected as modifications to the text reflected in the final report as appropriate.

Your opening statement addresses the variety of exposures and personal characteristics which were examined as potential risk factors. Among the associations you discuss are the results of our investigation of power line wire code configurations as surrogate indicators of residential 60 Hz EMF exposure. You state that our results are in opposition to the direction of relationships typically found in other studies.

Your statement is based upon results presented in the draft report pertaining to unmatched analyses of residential wire code comparisons for which both cases and controls could have resided in Woburn and upon a matched analysis of residences at diagnosis. These analyses include residences which were not the primary residences of interest regarding the assessment of EMF exposure and its relationship to childhood leukemia onset.

The most definitive analysis with regard to residential wire code is of the subgroup of persons who had what were considered stable residences, or cases and controls who resided in the same household for the entire etiologic period. These subjects would receive all residential EMF exposure in a single home and represent the best subset of the population to conduct the matched analysis. Results indicate an odds ratio of 2.20 which is consistent with the literature (London, et al. 1991, O.R.=2.15).

No association was identified between smoking during pregnancy and leukemia risk although findings of one study in the review article you cited demonstrates such a relationship. The preponderance of evidence to date has not upheld this relationship. Our

study allows for the generation of one risk estimate for the relationship between maternal smoking and childhood leukemia. Our research was not specifically designed to address such an issue and conclusions based upon a single risk estimate are not scientifically definitive. This uncertainty must be resolved by further investigations with the capacity to generate further associations to test the smoking and leukemia hypothesis.

Our confidence in our conclusions in this investigation are based on the strength of the exposure assessment associated with wells G and H and our ability to use our exposure risk estimate for a variety of refined analyses. The consistency in direction of the associations identified (Table 8 and 10) from these analyses allows our positive conclusions regarding potential for water exposure and childhood leukemia. Associations identified with other risk factors such as breast feeding and dental x-ray exposure (figure on page 3 of your critique) cannot be discussed with a level of certainty as no consistency in direction of association can be definitively established based on single risk estimates. Further, the total number of risk estimates calculated in this investigation would presuppose relationships identified as significant by chance alone.

Major Point 1 - Selection and Control of Confounders

This point is a critique of the methods and reasons we used for matching and selecting a comparison population. Your primary criticism is of the risk factors we controlled for in our analysis and the matching scheme we conducted in the course of control selection.

On page 4, paragraph 2 of your critique you state, "exposure to Wells G and H is likely to be evenly balanced in the study population." This statement is not correct. It was strongly suspected prior to the beginning of our study that potentially contaminated water was not available to all residences uniformly. The Murphy water distribution model (Final Report, Appendix XI) clearly demonstrated this fact. The location of the subject residences and the dates of residence during the wells operation had a direct bearing on how much contaminated water the residents potentially received.

It was absolutely essential therefore that the cases be matched to controls by age so that cases could be compared to controls who were consuming water during the same period of development at the same point in the timeline of the wells' operation. Potential exposure levels would depend on differences in water distribution patterns among residences over time as well as differences in concentrations of potential contaminants in wells G and H over time.

There were three times as many males as females in the case population. It was therefore decided to match by gender to assure that any gender-specific sensitivity of the subjects to potential exposures would be adjusted. Associations identified would not be gender-specific.

We also controlled for other confounders which we felt could be potential contributors to childhood leukemia onset. Some factors although not directly associated with leukemia are known to affect reproductive outcomes and were therefore included as part of the logistic regression.

Composite covariates were generated (Tukey, 1991) in an attempt to maintain statistical power while including major variables of concern. Although the Tukey analysis reduced the width of the confidence interval about the point estimates, small numbers of subjects continue to demonstrate the imprecision of the statistical calculations. Adjustment using the Tukey method does not change the direction or pattern of the identified associations. The magnitude of the risk estimates must be interpreted with caution.

In your critique, you point out that the direction of the shift in odds ratio values (Table 1, page 5 of your comments) is not consistent for each segment of the etiologic period. You then associate the variability in direction with bias. You have not considered that the etiologic period for each case-control triplet differs in length. The crude analysis for the full etiologic period allows study subjects with different lengths of exposure to be compared directly. As a result, persons who were younger at diagnosis, who logically will be more likely to have a smaller cumulative exposure are compared to those with longer exposures. Crude analysis for the period from birth to diagnosis also has the same limitation.

Crude rates are composed in part of comparisons between persons of differing ages with differing exposure simply because they lived for different lengths of time in a potentially exposed population and not because they were necessarily at greater risk of exposure. Crude risk estimates therefore, should be interpreted with caution particularly considering the availability of matched results.

The crude analyses for the period during pregnancy is by definition an analysis with defined time limits based on the beginning and end of the pregnancy. Exposure comparisons during these periods would be limited to more well defined periods of exposure and not have the same limitation as the other crude analyses.

In the matched analysis, cases are compared to their age and date of residence matched controls. They are at risk of having an exposure of the same maximal length of time during the same period of time of the Wells' operation. Differences in exposure values reflect differences in the public water system's delivery of the contaminated water, not differences in well contaminant concentrations. Adjusted values give the best comparison between exposure levels of individuals residing in Woburn.

The size and direction of the risk estimates of potential confounders are based on risk estimates of very small samples. This study cannot therefore fully address the effect of each potential confounder on outcome. Our conclusions regarding wells G and H exposure are based on our study's ability to compile a body of evidence which addresses criteria for causal inference including consistency in direction of associations identified, a

dose-response relationship, and a decrease in risk after the removal of the pathway of exposure.

Our inclusion of a variety of different variables in the model was done to explore the potential unexplained confounding which may take place. We were consistent in presenting unadjusted odds ratios as well as those of adjusted rates. The unadjusted matched analysis can be seen to demonstrate the same trend in association as the adjusted between exposure to water from wells G and H and childhood leukemia risk.

The comments presented herein substantiate the necessity to use matched controls based upon age, sex, and residence in the Woburn analysis. The unadjusted conditional logistic analysis using matched data does not therefore overestimate the risk. No matter the preference of the reader regarding risk estimates, the association between the potential for exposure to water from wells G and H and childhood leukemia is positive and greatest for the period during pregnancy.

Major Point 2 - Inconsistency with Lagakos *et al.* (1986)

With respect to the comparison of our study conclusions to those of Zelen and Lagakos, your point is well taken that our conclusions are not in fact similar. The associations Zelen and Lagakos identify between potential for exposure to the wells and childhood leukemia are for the time period from birth to case diagnosis. They discussed no association between exposure during pregnancy and leukemia diagnosis as we did in our analyses.

You stated that "Lagakos *et al.* (1986) found an apparent association between exposure to wells G and H from birth to case diagnosis, where as this study found no such association." You further state "the results of the Lagakos study and the DPH study are therefore inconsistent, although both studied essentially the same set of cases and both ascertained exposure to Wells G and H during this same etiologic period by very similar methods." This is not correct.

Zelen and Lagakos present no results specific to the pregnancy period. When one examines their method of assigning exposure values it seems that they include the pregnancy as part of the Birth to Diagnosis exposure time period. They state (pg. 585-586) "we merged Wells G and H exposure information with other data and assigned to each pregnancy the annual exposure score corresponding to the mother's residence in the year the pregnancy ended."

Zelen and Lagakos computed yearly exposure values based on five zones of exposure in Woburn. They compared the yearly values for each study subject to yearly exposure values of other children in the same residential areas. These values were calculated based on the exposure average and their variance for children born in the same year who were not diagnosed with leukemia. Based on their description of their exposure assessment

method Zelen and Lagakos were most likely unable to refine their exposure assessment enough to specifically detect the relationship during pregnancy.

Our exposure data was based on aggregate totals of month by month exposure values for each case and their controls. We were thus able to sum monthly exposure values based on where the mother lived relative to more than fifty zones of exposure during each month of exposure in Woburn. This allowed for the generation of exposure values specific to the months of the pregnancy and the residence or residences the mother lived during the pregnancy.

The effect Zelen and Lagakos identified may represent at least part of the relationship we identified between exposure during pregnancy and leukemia. The exposure values they generated are not specific enough to identify exposures during pregnancy alone. Zelen and Lagakos assigned values based on the year the individuals were born. This type of assessment would assign a mixture of exposure values from the year of conception and the year of birth. Children born early in the year (12 of the 21 cases were born on or before the 6th month of the calendar year allowing for a large amount of misclassification error) would be assigned an exposure value representing that year. More than one third of the cases would have been assigned an exposure score representing the calendar year after they were conceived. This means that the actual exposure during the first trimester of pregnancy (potentially the trimester for which the child is most sensitive to environmental exposures) would not have been considered in the assignment of an exposure score. Also, the observational analyses (summary statistics) used by Zelen and Lagakos do not consider short term extremes of exposure which may have contributed to the association with illness, particularly during specific developmental periods of pregnancy.

Major Point 3 -Inclusion of a lymphosarcoma/leukemia case

Point 3 of your review is specific to the concern that our study included a leukemia case which was originally diagnosed as a Lymphosarcoma that later became a leukemia. This child is in fact included in our study. The lymphosarcoma/leukemia subject fits our case definition criteria as described in the methods section of our report. We do not have exclusion criteria for cases based on pre-existing health conditions as part of our case definition. The case therefore was correctly included as part of our study population.

Major Point 4 - Rationale for case eligibility dates

Case ascertainment ended as of August 31, 1989. As discussed in the final report, inclusion of the cases diagnosed since that time would not have changed the conclusions of this investigation. In all instances the new cases were too young for either maternal or personal exposure to water from the contaminated wells during the etiologic period of this investigation.

The researchers who conducted the MDPH 1981 Childhood Leukemia Study selected January 1, 1969 as the earliest date of diagnosis for which a case could be included in this investigation. They determined that cases diagnosed prior to that time could not be located in a reliable manner leading to the potential for underestimation of leukemia incidence prior to that date.

Major Point 5 - Matching

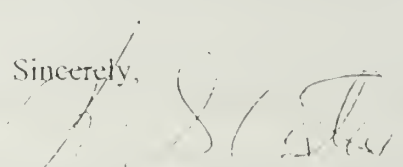
The matching procedure as previously explained contributes to the precision of the case-control analysis. The water distribution model by definition assigns values of zero to cases or their matched controls if they are not receiving water from wells G and H. If the wells were functioning at a point in time, then a case or matched control might receive some or no exposure from the wells. If the wells were closed then cases and controls are all assigned a value of zero exposure and are not part of the analysis for the portion of the etiologic period in which they receive no exposure. As in any analytic adjustment procedure, stratification of the data allows for the examination of specific subjects of a population that share a common characteristic.

"Adjusting" for the effect of that characteristic provides information regarding how the association in question behaves in the known presence or absence of the characteristic. In this situation we have stratified the analysis to include matched triplets (a case and two controls) for whom at least some member received some exposure during pregnancy. We are deliberately eliminating triplets who all received no exposure during pregnancy (concordant triplets) and then determining if exposures were likely to be associated with outcome among the remaining subjects (discordants).

For the purpose of this analysis, the population is reduced to the persons who were in-utero and who received at least some exposure. Although statistical power is reduced, the analysis allows for the assessment of potential exposure during pregnancy independently of potential exposure to mother and child at any other time.

I would like to thank you for taking the time to review our work and provide constructive comments.

Sincerely,



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